SURGERY

VOL. 4 NOVEMBER, 1938 NO. 5

Original Communications

THE TREATMENT OF PARALYTIC BLADDER IN CASES OF SPINAL CORD INJURY*

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INTRODUCTION

THE ideas which follow have not come through trial and error. Proof by my own hands of the merit of any method is wanting. In this sense is your choice of me for this address inapt. A study of the reports of those with experience, however, discloses such a difference of opinion on just what should be done for the bladder in spinal injuries that my inexperience may fit me as well as another to the task of putting the facts truthfully before you.

The problem is urological, and anyone trained in urology and so minded can find in these facts the points on which our best authorities disagree. Perhaps careful analysis will disclose to those in need, like myself, a rule of accord to serve on the rare occasion as a safe guide in the care of paralyzed bladders. This care never was a matter for general orders in any army during the World War and how much the disservice of casual treatment accounts for the death within a few weeks of 80 per cent of the 2,324 soldiers in our army who suffered injury to the spinal cord is worth pondering. Many of these men would have recovered from their nerve injuries, completely or partially, had they lived. Mostly they died, not from the injury to the spinal cord, but from the urinary complication. Although in civil life automobiles and not bullets cause most of the injuries to the spinal cord, the problem of care of the bladder is the same as in an army at war. While fatalities are fewer, they mostly follow the same complications: namely, urinary infection and bedsores on skin areas continually soiled by urine. The magnitude of the problem in every injury of the spinal cord is evident.

*Read before the American Academy of Orthopaedic Surgery, Los Angeles, Calif., Jan. 19, 1938.

Received for publication, June 9, 1938.

One wonders to what extent the high death rate is the result of neglect of the bladder-problem through failure to realize its importance; to what extent it follows lack of knowledge and skill in the use of the methods of care, although those methods were properly chosen at the time; and to what extent it results from poor judgment in the choice of method. All of these factors may be variably responsible. The last factor implies a choice and it is on this that authorities differ. The three methods most widely advocated are: First, noncatheterization and manual expression; second, suprapubic cystostomy; and third, retention catheter.

Before discussing the relative merits of these methods, and of others which are less popular, the urological features peculiar to the problem should be made known, because a comparison of values rests largely on an understanding of this particular aspect. Authorities fail to mention this point which appeals to me as distinctively central. Perhaps each failure, as well as each success, can be accounted more to manner than to method, for, when faultlessly executed, one method may be as good as another. If this be true, the best way to treat the bladder will vary with circumstances and conditions.

The musculature of micturition and its innervation are complex and not fully understood. There are five groups of muscles innervated by three main pathways, as follows:

Smooth Muscles	*Autonomic Nerves*
1. The detrusor vesicae	I. The hypogastric pathway Thoracicolumbar sympathetic system
2. Internal sphincter	
3. Musculus trigonalis	II. The pelvic pathway Sacral parasympathetic system
Striated Muscles	*Somatic Nerves*
4. External sphincter 5. Accessory urethral and perineal muscles	III. The pudendal pathway

There are five regulating centers of micturition, namely:

1. Cerebral	Voluntary impulses
2. Subcortical 3. Lumbar 4. Sacral 5. Peripheral ganglionic plexuses	Automatic reflexes

The voluntary impulse originates in the brain and is delivered by the midbrain to the spinal centers, instituting various nervous reflexes into automatic action. Through the hypogastric nerves from the lumbar sympathetic center flash inhibitory signals to the detrusor, and stimulating impulses to the sphincter; whereas, through the pelvic nerves from the sacral-parasympathetic center, reciprocally antagonistic impulses flow, stimulating the detrusor and inhibiting the sphincter. These

reflex impulses show an integrative activity not unlike peristalsis. The trigonalis muscle has sympathetic innervation only, no parasympathetic, so that, with sympathetic inhibitory impulses to the detrusor, flow stimulating impulses to the trigone. The striated muscles receive voluntary impulses through the pudendal nerve.

The mutual antagonism of the foregoing autonomic systems is only partial. One set does not become dominant with loss of the other. An intact parasympathetic system is more essential to function than an unbroken sympathetic pathway. In fact, hypogastric innervation may be destroyed at the periphery with little disturbance of micturition. All depends on the level, peripheral or cortical, at which the break occurs. Voluntary micturition arises from the brain, involuntary micturition from the cord, and complete dissociation gives an automatic bladder, variations of which are explained by the mutual antagonism of the sympathetic and parasympathetic systems in relation to the level at which the dissociation is made. If this level lies above the lumbar center, automaticity will be good and may be influenced reflexly by sensory impulses. Normally, tickling the sole of the foot will cause withdrawal of the foot. In the absence of this response, there is an overflow of motor impulses to other structures and it is this so-called mass reflex which causes the bladder to contract (Mayo-Robson, Kidd, Head and Riddoch). According to Holmes, the sudden increase of intra-abdominal pressure by augmenting tension within the bladder causes it to contract. He was led to this explanation by observing that contractions of the bladder always followed the abdominal spasms "by a definite interval of time, sometimes by several seconds." If the lesion in the cord is below the lumbar center, automaticity will be poor or absent and stimulation of the soles of the feet now may fail to cause this abdominal reflex and thus have no effect. Retention may follow either irritation of hypogastric fibers from the lumbar center or paralysis of the pelvic fibers from the sacral center, and incontinence comes, vice versa, with irritation of the pelvic or paralysis of the hypogastric fibers. It would be expected, therefore, that the effect on micturition will vary considerably with the level of the injury to the spinal cord. It will vary also with the type of injury. A complete transverse lesion will have a more constant and lasting effect than a partial tear—as from the laceration of a fragment of bone, or an injury without rupture of any nerve fibers—as from hemorrhage or edema with pressure on the cord. Injuries in the region of the cauda equina are notable for the marked variation in effect.

A. The Effect on Micturition.—Three phases describe the common effect on micturition of a transverse lesion at any level of the cord or cauda equina. These are: first, retention; second, overflow incontinence; and third, automatic micturition. The first phase is constant for all lesions, though variable in duration. In fractures below the

lumbar center, the other effects may be slight or absent. The phase of urinary retention commonly lasts from two to three weeks but may disappear within twenty-four hours or persist eighteen or more months. Retention, in the presence of injury at any level, is explained on the basis of "spinal shock" which isolates the bladder from its reflex centers. Complete paralysis of the detrusor and spasm of the sphincter result. Because of this spasm, catheterization usually is difficult. The picture of vesical relaxation and atony is seen cystoscopically and cystometric records all show an inert vesical wall with no "stretch" response. Reflex contractility is entirely absent. Unless infected or injured, these distended bladders never rupture spontaneously nor is there any record that the back pressure of distention ever produces hydronephrosis and renal insufficiency.

The second phase, an overflow incontinence, is of short duration and may be poorly pronounced. The detrusor gradually recovers tone, without the power of contraction however and, along with the recovery, a gradual weakening of the spasm of the sphincter occurs. In consequence, urine dribbles out as an overflow incontinence. An inlying catheter may be repeatedly extruded and, even when retained, urine may leak around it. This may be the final phase after destructive lesions of the sacral segments or cauda equina.

The third phase is the development of an automatic bladder. With the gradual gain in muscle tone, contractility slowly returns to the detrusor and its muscles hypertrophy. Along with this recovery of the detrusor there is a return of the sphincteric spasm so characteristic of the first phase. The insertion and retention of a catheter again become more difficult. A gradual recovery of the normal reaction to "stretch" is shown by cystometrograms, and the condition of automaticity develops. Automatic micturition occurs sooner and more perfectly after fractures above the lumbar center than after those below, but I can find no record of it after divisions of the cauda equina. It differs from normal micturition in the incoordination of detrusor and sphincter. Instead of being reciprocal, their activity becomes synchronous. With the return of its tone and contractility, the detrusor, in order to establish automaticity, must work against the unwonted coincidental contraction of the sphincter, and, with good automaticity, it will hypertrophy and the bladder will return to normal dimensions. So high may this initial return of tonus become, sometimes, that, unless preventive measures are instituted, the capacity of the bladder will be reduced to almost nothing. The smallest amounts of urine set up emptying contractions so that the patients are as wet as in the previous phase, but for another reason. Should the detrusor fail to hypertrophy at this stage, as is likely with infection or severe toxemia, there can be no automaticity. Overdistention will persist. The detrusor will then become more and more atonic and an overflow incontinence will occur or

simply continue from the second phase. Or the condition may revert to that of retention as in the first phase. In conditions between these extremes of hypertrophy and atony of the detrusor, partial and atypical forms of an automatic bladder may occur.

The usual course of the effect on micturition of a transverse lesion of the cord at any level, therefore, is first, retention; second, overflow incontinence; and third, automaticity. These phases are most constant with complete transverse lesions above the lumbar center but, even then, are variable in duration. Variations from this course, both as to phase and duration, occur commonly with transverse lesions below the lumbar center, with partial divisions or less destructive lesions at any level, and with the advent of certain complications, such as urinary infection. True paralytic incontinence, which differs altogether from the usual picture of cord bladder, sometimes follows destruction of the lumbar centers. Automaticity never develops with destruction of sacral segments or cauda equina. With some partial lesions or injuries, there may be only delay in starting micturition, the stream may be without force and dribbling may occur at the end. In such cases normal micturition usually is soon established. The midcervical and dorsolumbar segments of the cord are injured most frequently, and therefore the three successive phases of effect on micturition are the commonest. It is well in each case to surmise, if possible, the neuromuscular background of each phase of effect in relation to the level of the cord injured and to the type of injury in the selection of any particular method of treatment. It must be obvious to all that the problem of treatment is distinctively urological.

B. The Treatment.—The information in the literature is inadequate as a guide to what should be done for the bladder in injuries of the spinal cord. There seems to be an even choice between three methods; namely: (1) noncatheterization and manual expression, (2) suprapubic cystostomy, (3) retention catheter with or without tidal irrigation.*

Each method has its particular advantages and disadvantages; each receives as much condemnation as approbation, and each has just as valid reasons against its choice as for it. The dissenting authorities are as reliable as the assenting ones. Naturally, when called to work in an open field of this kind, one who has no row of his own to hoe will hesitate which one to choose. Upon two points, however, all those with experience agree; namely, no paralyzed bladder should be left alone to distend and overflow, and, the urethral catheter should never be used casually or intermittently. Possibly this seeming paradox holds the solution to the question of a choice of method. It illustrates at least the urological aspect of the problem, and to my mind this *is* the problem. "The first and fatal catheter" receives sufficient castigation in the literature. On the other hand, neglect and overdistention may be fully as criminal

*The literature has been summarized in an appendix by Dr. Henry M. Weyrauch, Jr.

from the urological standpoint. Overdistention is extremely dangerous in the presence of infection, and overflow of urine means bedsores. The purpose of all treatment is to control infection and prevent bedsores. The success of any method directed to this aim, which may have to be carried on for weeks and months, depends largely on the efficiency and reliability of the personnel which will conduct it; on the nurses, relatives, orderlies, or friends to whom goes the routine of emptying the bladder by manual expression or the care of catheters and irrigations. The urologist knows that this routine care must be precise and regular. Foreknowledge that it may be wanting should influence the choice of that method least dependent on routine. But failures cannot always be assigned to mismanagement. Environment and manner of conduct are but a part of the urological problem. Patients vary in their response to method. Some tolerate a catheter poorly. Persistence in its use in the presence of urethritis, prostatitis, or epididymitis also shows poor judgment. Therefore, any precise and regular routine should still be flexible and open to change at need.

The other part of the urological problem is the condition of the bladder itself; that is, the particular neuromuscular disturbance which demands treatment. The phases of change in the disturbance of micturition can be predicted only partly by knowing the level of the injury to the cord. The succession from retention, through overflow incontinence, to final automaticity after lesions above the lumbar center may be retarded or interrupted, either partly or completely, by intercurrent infections or toxemia. Any operation for the repair of the broken back at any period of this succession may throw the condition back to the initial retention. The purpose of noncatheterization and manual expression is to hasten the development of an automatic bladder. Patients with good automaticity get along very comfortably. One side of the question then is whether this method should ever be used for patients with lesions below the lumbar center, after which automaticity is known never to develop. This is a difficult question. Retention is the initial phase for all lesions irrespective of type and level. It may not be possible to estimate immediately the extent of the lesion or its exact level and whether or not automaticity will develop. The injury, in spite of appearances, may be mild and the patient may recover quickly. The other side of the question is whether cystostomy or insertion of a catheter ever should be immediate. A catheter, whether urethral or suprapubic, means infection. It is true that this infection usually can be controlled. This, however, may be difficult. On the other hand, should urinary sepsis develop spontaneously, it may then be too late to save the patient by drainage of any kind.

The three methods grade themselves from minor to major procedures. The conservative course on patients seen immediately after injury would

be to apply them in this order, changing from minor to major according to particular indications as they arise.

On a patient seen early one would first try to empty the bladder regularly by applying pressure with the open hand above the symphysis. In doing this the bladder should not be squeezed. Should it have become too distended by delay, manual expression may cause extreme pain which may be relieved by morphine. Having the patient lie on his side may be of help, as will also pressure on the bladder through the rectum. The method works better on women. It should be applied regularly every six to eight hours, and the bladder never should be allowed to overdistend. Too prolonged an interval between expressions, by allowing overdistention, may throw the bladder back into atony with a loss of all that has been gained; but even more important than this is the likelihood that infection may get a start by reason of this neglect. The advent of infection is the one real contraindication to the use of this method. It should be looked for daily. It is dangerous to use manual expression in the presence of infection. Ascending pyelonephritis is likely and bladders have been ruptured. For a patient with clear urine, routine manual expression is safe and may lead to the rapid development of an automatic bladder. It is not the ideal procedure for all patients, nor will it always work. When infection mars its usefulness, when it fails for some other reason, when the patient has already had his bladder emptied by catheter, or when the neuromuscular phase is other than retention and unsuited to its use, one of the other two methods should be adopted promptly.

The use of a retention catheter has few advocates compared to suprapubic cystostomy. Urologists know something of the continuous care and attention required for a retained catheter. If this expert and faithful assistance is available, a retention catheter may save time, control the infection and keep the patient dry and comfortable until such time as automaticity develops. It is well to state again the fact that all authorities condemn intermittent catheterization. Recently Munro* has strongly advocated the use of tidal irrigation and claims by it to have reduced infection from 72 per cent to 14.2 per cent. Furthermore, tidal irrigations stimulate the bladder to regain contractility and therefore hasten the development of automaticity.

Suprapubic cystostomy has many advocates, most of them recent. The method gives good drainage. The care of a suprapubic tube is simple compared to that of a retention catheter. Tidal irrigations may be given through it or suction applied on occasion. Upon considering the many months that drainage may be required, the suprapubic tube has distinct advantages over the retention catheter. One possible objection is persistence of a suprapubic fistula upon development of an automatic bladder.

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SUMMARY

I. It is judicious to treat each paralyzed bladder on sound urological principles.

A. There are four effects on micturition to consider: retention, overflow incontinence, automaticity, and true incontinence. In cases of fracture above the lumbar center the first three occur in succession. An automatic bladder never develops as the late and final effect after fractures below this center, and incontinence may follow destruction of the cord at this level. Partial lesions and trauma by pressure or bruising effect slight changes from the normal, usually of short duration. The "spinal shock" of any lesion at any level causes an initial paralysis of the bladder with retention. Early development of automaticity is highly desirable, but it is possible in only one group. Some methods favor this outcome more than others. Therefore, the same indication for the treatment of retention at onset notwithstanding, consideration of the probable late effects presumed by a knowledge of the level and extent of the injury should influence the choice of method.

B. Any method proves itself largely by reason of the precision and skill with which it is carried out. Some methods require more care and experience than others. Consideration of the fact that this care may be wanting also should influence the choice of method.

II. It is impractical to treat all paralyzed bladders alike.

A. Some do best if left alone or, at most, emptied regularly by manual expression. Others get along satisfactorily with retention catheters irrigated periodically or continuously (tidal); and still others require cystostomy for drainage.

B. Urinary infection and bedsores foil the outcome to a maximum of over 80 per cent. The intent of all care, therefore, is to prevent or control these two evils.

C. Noncatheterization, with manual expression and antiseptic therapy, is the one way to ward off urinary sepsis. When it works, it is the best method to hasten automaticity. It is useless and dangerous as a method to control infection. If infection is present, the choice between catheter per urethram, and cystostomy must be made at once. Even before infection takes place drainage by one means or the other often proves superior.

III. The care of the bladder after injuries to the spinal cord should be prompt, precise, and farsighted. To be successful, rare judgment and constant attention are required. Most of these patients, in spite of all effort, die of or with severe urinary infection.

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Supplementary Bibliography

I. BIBLIOGRAPHY OF THE PRINCIPLES OF TREATMENT

A. *Noncatheterization and the Use of Manual Expression.*—

Advocated by: Heddaeus,[42] 1888; Chico,[15] 1889 (who practiced it in 1874); Besley,[5] 1917 (experience in Base Hospital, France); Vellacott and Webb-Johnson,[83] 1919 (experience in British war hospital); Plaggemeyer,[72-74] 1919, 1921 (experience with American army in World War); David,[22] 1921; Cumming,[19] 1922 (United States Army); Cahill,[13] 1928; Gurdjian,[35] 1931; Connors and Nash,[17] 1934 (54 cases).

Reasons for: 1. Prevents urinary tract infection.
2. Best method for the rapid development of an automatic bladder. Many patients with spinal injury can be saved if they do not succumb to urinary tract infection. By this method there is no damage to the kidneys and there is no danger of spontaneous rupture of the bladder.

Method condemned by: Kidd,[48] 1919; Boyd,[9] 1929.

Reasons against: 1. Hematogenous infection will result if patients are not catheterized.
2. Method is time-consuming in way of nursing care.
3. Urine will soil bed and cause bed sores.

B. *Retention Catheter.*—

1. *Without Tidal Irrigation.*—

Advocated by: Kidd,[48] 1919 (war experience); Gurdjian,[35] 1931 (if infection is present).

Reasons for: 1. It saves time.
2. It avoids overdistention of the bladder.
3. It reduces infection to a minimum.
4. Skin is not soiled by urine; prevents bed sores.
5. Calculi do not form.
6. Urethra not damaged by frequent catheterizations.
7. Paves the way to an automatic bladder.

Condemned by: Drapier,[26] 1919; Connors and Nash,[17] 1934.

Reasons against: 1. Invariably leads to infection.
2. Not effective method of drainage; frequent clogging of catheter from clots.

2. *Retention Catheter With Tidal Irrigation.*—

Advocated by: Munro,[70] 1937 (has reduced urinary tract infection from 72 per cent to 14.2 per cent); Young,[91] 1926 (recommends decompression apparatus with mild antiseptic).

Reasons for: 1. Prevents overdistention of bladder.
2. Reduces infection.
3. Facilitates development of an automatic bladder.

C. *Suprapubic Cystostomy.*—

1. Early.
2. In presence of urinary tract infection.

Advocated by: Thompson Walker,[80] 1917 (no experience with method advised); Mayo-Robson,[63] 1917; Drapier,[26] 1919; Wertheimer,[88] 1924 (experience in French army); Boyd,[8, 9] 1927, 1929; Wallenstein,[86] 1931; Edwin Davis (personal communication to Munger,[68] 1937); Deming (personal communication to Munger[68]), 1937.

With exception: Esau,[29] 1921, if severe bladder infection; David,[22] 1921, as a last resort; Conners and Nash,[17] 1934, if expression painful or bladder overdistended; Creevy,[18] in selected cases; Munger,[68] 1937, if expression method is unsatisfactory.

Reasons for: 1. Prevents overdistention of bladder.
2. Prevents severe infection of bladder.
3. Prevents ascending infection.
4. Permits adequate drainage (large tube, simple to irrigate).
5. Economical in time of nurses.

The suprapubic fistula will heal and an automatic bladder develop, if desired. Hematogenous infection of urinary tract will result even though the patient is not catheterized. Continuous bladder irrigation may be instituted if so desired.

Condemned by: Kidd,[48] 1919; Vellacott and Webb-Johnson,[83] 1919; Young,[91] 1926.

Reasons against: 1. Difficult to care for from nursing viewpoint.
2. Severe infection of the urinary tract following use of the method is the rule rather than the exception.
3. Bladder calculi likely to form.
4. If automatic bladder develops, practically impossible to get fistula to close.
5. Difficult to keep drainage tube water-tight (leakage causes bed sores).
6. Bladder is permanently damaged.

D. *Intermittent Catheterization.*—
Advocated by: None.
Condemned by: Drapier,[26] 1919; Kidd,[48] 1919; Plaggemeyer,[72, 73] 1919; Curtis,[21] 1919; David,[22] 1921; Cumming,[19] 1922; Young,[91] 1926; Cahill,[13] 1928; Boyd,[9] 1929; Connors and Nash,[17] 1934; Wesson,[89] 1934.
Reasons against: 1. Traumatizes urethra.
2. Causes prostatitis, seminal vesiculitis, and epididymitis.
3. Invariably results in urinary tract infection.
4. Fails to keep bladder empty; danger of overstretching wall.
5. Leakage between catheterizations predisposes to bed sores.

E. *Distention and Overflow.*—
Advocated by: None.
Condemned by: Vellacott and Webb-Johnson,[83] 1919.
Because: 1. Pain may be severe.
2. Distention of bladder may embarrass respiration and cardiac action, an important factor in hemothorax.
3. Shock may be induced by allowing distention of bladder.
4. Doubt whether bladder will ever regain tone.
5. Hematuria, vesical in origin, may ensue.
6. Extremely dangerous if infection is present.

F. *Suprapubic aspiration* in preference to catheterization if intervention is necessary.
Advocated by: Plaggemeyer,[72, 73] 1919.
Reason: Will avoid infection.

G. *Presacral Sympathectomy.*—
Advocated by: Learmonth,[58, 59] 1931; McCrea and Mcdonald,[65] 1934; Creevy,[18] 1936; Grant,[33] 1936 (reports case following spine injury; patient died).
Condemned by: Braasch,[10] 1934.

H. *Instrumental Dilatation of the Urethra.*—
Advocated by: Vellacott and Webb-Johnson,[83] 1919.
Reason: Produces temporary incontinence until automatic bladder develops. No cases.
(Braasch[11] advocated method in atonic bladders, 1934.)

I. *Nerve Transplantation to Paralyzed Bladder.*—
Trumble[82] (experimental), 1935.

J. *Sinusoidal Stimulation of Bladder.*—
Cumming,[19] 1922 (serious complications in 2 cases; no details).

Supplementary Forms of Treatment.—
1. Urinary antiseptics.
2. Drugs which act through sympathetics and parasympathetics.
3. Drugs acidifying urine to prevent calculus formation and reduce infection.
4. Prostatic massage and urethral dilatations to reduce obstruction.
5. Bladder irrigations to reduce infection.
6. Instillation of irritating drugs to cause bladder to regain tone.
7. Autogenous vaccine to combat infection.

II. NEUROANATOMY, PHYSIOLOGY, AND PATHOLOGY OF MICTURITION

Dubois,[27] 1876; Mosso and Pellacani,[66] 1882; Born,[7] 1886; Guyon,[37-39] 1889, 1891, 1900; von Zeissl,[84, 85] 1896, 1901; v. Frankl-Hochwart,[31] 1898; Elliott,[27A] 1907; Schwarz,[78] 1916; Fearnsides,[30] 1917; Head and Riddoch,[41] 1917; Thompson Walker,[80] 1917; Adler,[1] 1918; Muller,[67] 1918; Stavianiček,[79] 1918; Barrington,[3, 4] 1921, 1933; Langley,[50] 1921; Elsberg,[28] 1926; Potter,[75] 1926; McClintic,[64] 1928; Beattie,[6] 1930; Gurdjian,[35] 1931; Learmonth,[59] 1931; Denny-Brown,[24, 25] 1933; Holmes,[45] 1933; Gruber,[34] 1933; Karschulin,[47] 1933; Langworthy,[51-54] 1933, 1934, 1935; Connors and Nash,[17] 1934; Grant,[33] 1936; Lewis and Dees,[61] 1936.

III. EFFECT OF LEVEL OF CORD INJURY ON MICTURITION

Thompson Walker,[80] 1917; Adler,[1] 1918; Plaggemeyer,[72, 73] 1919; Barrington,[3] 1921; Elsberg,[28] 1926; Denny-Brown,[25] 1933; Holmes,[45] 1933.

IV. PHASES IN BLADDER DISTURBANCE FOLLOWING CORD INJURY AND EFFECT OF SEVERE TOXEMIA

Head and Riddoch,[41] 1917; Thompson Walker,[80] 1917; Plaggemeyer,[72, 73] 1919; Vellacott and Webb-Johnson,[83] 1919; Denny-Brown,[25] 1933; Holmes,[45] 1933; Munro,[70] 1937.

V. CYSTOSCOPIC PICTURE

Caulk,[14] 1919; Plaggemeyer,[72, 73] 1919; Cumming,[20] 1932; Lewis and Dees,[61] 1936.

VI. ETIOLOGY AND CONSIDERATION OF INFECTION OF URINARY TRACT SECONDARY TO SPINAL CORD INJURY

Thompson Walker,[80] 1917; Curtis[21] (experimental), 1919; David[22] (experimental), 1921; Boyd,[9] 1929; Cumming,[20] 1932; Connors and Nash,[17] 1934.

VII. CONSIDERATION OF HEMATURIA

Vellacott and Webb-Johnson,[83] 1919.

VIII. EFFECT ON KIDNEYS OF CORD BLADDER SECONDARY TO SPINE INJURY

Curtis[21] (experimental), 1919; Cumming,[19, 20] 1922, 1932; Cahill,[13] 1928; Hepler,[44] 1933.

IX. EFFECT ON URETERS

Fullerton,[32] 1919; Kreutzmann,[49] 1928.

X. RECTAL SPHINCTER PARALLELS URINARY

Plaggemeyer,[74] 1921; Creevy,[18] 1936.

XI. SEX FUNCTION (IN CORD INJURY)

Plaggemeyer,[74] 1921; Cumming,[19] 1922.

XII. PRIAPISM (FOLLOWING CORD INJURY)

Hartwell,[40] 1917.

XIII. FOLLOW-UP RESULTS—STATE OF AUTOMATIC BLADDER

Plaggemeyer,[74] 1921; Cumming,[19] 1922; Hepler,[44] 1933.

XIV. PROGNOSIS

Drapier,[26] 1919; Connors and Nash,[17] 1934; Creevy,[18] 1936.

TUMORS OF THE KIDNEY WHICH INVADE THE INFERIOR VENA CAVA

A Report of Seven Cases

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(From the Mayo Foundation and the Division of Medicine and the Section on Pathologic Anatomy, the Mayo Clinic)

INVASION of the venous circulation by malignant tumors which originate in the kidney occurs with surprising frequency. Judd and Scholl in a study of 200 renal tumors found that 22.5 per cent had invaded the renal vein. More extensive invasion of the venous circulation with involvement of the inferior vena cava occurs much less frequently. Excellent studies of case reports of this type of lesion have been made by Pleasants, Simpson, Jacobson, and Goodpasture. Simpson in 1924 collected 32 cases in which a renal tumor had involved the inferior vena cava. Two additional cases have been reported since that time; these cases and the 7 cases included in this report bring the total to 41.

Investigators of this subject have complained that the nomenclature of renal tumors in older reports interferes with an accurate classification of the type of tumor. It is probable that recent literature has merely added to this confusion for two reasons: first, the controversy over the Grawitz theory has led to the use of a number of different names for the so-called hypernephroma; and second, the well-known polymorphism of this type of tumor has led many authors to classify peculiar examples as "perithelioma," "sarcoma" or "endothelioma." However, at the Mayo Clinic we usually refer to the Grawitz tumor as "carcinoma" and will so designate the seven tumors which form the basis of this report.

It is apparent that nearly all types of malignant renal tumors are capable of invading the renal vein and inferior vena cava. This was demonstrated by Engelken and Rosenstein, who have reported cases in which such involvement occurred; in these cases the patients were children and the tumors were sarcomas. Taddei reported a case in which the tumor was a squamous-cell carcinoma of the renal pelvis, and Turner and Velpeau have reported cases in which the renal tumor was a metastatic growth.

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Received for publication, June 20, 1938.

In the 34 cases of tumorous invasion of the inferior vena cava reported previously, the tumors occurred predominantly among males (28 cases) and occurred more often in the right kidney (20 cases). It was suggested by Pleasants that the shorter renal veni on the right side may possibly give the tumor easier access to the inferior vena cava. The tumorous thrombosis extended into the right auricle in 16 of the 34 cases.

Symptoms are often obscure and it is apparent that diagnosis is usually either established or completed at necropsy. Edema or ascites or both occurred in 24 of the reported cases, and pain of varying degree, character, and situation occurred in 21 cases. The presence of the most helpful indication of obstruction of the inferior vena cava, namely, engorgement of the collateral superficial veins, was noted as present in only 11 cases. However, the absence of this sign was noted in 13 cases. Hematuria, which is a helpful indication of renal tumor, was noted as present in 10 cases and as absent in 11 cases. Its presence or absence was not indicated in 13 cases.

Comments concerning the collateral circulation that is quickly established when the inferior vena cava is obstructed have been made by both Simpson and Pleasants. It is indeed amazing that compensation for complete obstruction can occur with such a paucity of physical signs. In several reported cases obstruction of the hepatic veins has occurred and has produced icterus. In one case reported, obstruction of the portal vein also was present. Woodruff and Levine have suggested diagnostic methods which may be of value in establishing the presence of venous obstruction. None of these procedures was used in our cases. Unfortunately, in some cases either tumorous thrombosis or a superimposed simple thrombosis descends into the iliac veins. In such cases these methods probably would not enable one to make a positive diagnosis of obstruction of the inferior vena cava. However, because of the high incidence of tumorous thrombosis in cases of obstruction of the inferior vena cava (24.8 per cent, according to Pleasants) and owing to the high incidence of renal tumors in cases of tumorous thrombosis (50 per cent, according to Simpson), the use of these methods is suggested in cases in which a renal tumor is suspected, but the symptoms are obscure. Sudden death sometimes occurs in cases in which tumorous thrombosis involves the heart. In such cases death is caused by obstruction of the tricuspid valve or by pulmonary embolism resulting from the tumorous thrombosis or the superimposed simple thrombosis.

REPORT OF CASES

CASE 1.—A white man, aged 56 years, came to the clinic because of ascites and edema of the legs. The initial symptom had been a mild edema of the feet which had occurred at night. This symptom first had been noted five months before the

patient came to the clinic. Examination had disclosed that the liver was enlarged. A week after the appearance of the edema, thrombophlebitis of the right leg had developed; the patient had been confined to bed for seven weeks. Four months before he came to the clinic, severe hematuria had occurred; the severe hematuria had lasted only one day and the blood in the urine had disappeared promptly. After this the edema of the feet had improved but had not disappeared entirely. Three weeks before the patient came to the clinic ascites had developed; this had increased in severity and had caused a gain of 23 pounds (10.4 kg.). At the same time the edema of the legs had become much worse than it had been. Ten days before he came to the clinic, 2,000 c.c. of ascitic fluid had been removed by abdominal paracentesis.

Examination at the clinic disclosed definite ascites. The superficial veins of the abdomen were moderately dilated. There was moderate edema of the genitals and over the sacrum. There also was marked edema of both legs. The liver was palpable 10 cm. below the right costal margin. The concentration of hemoglobin was

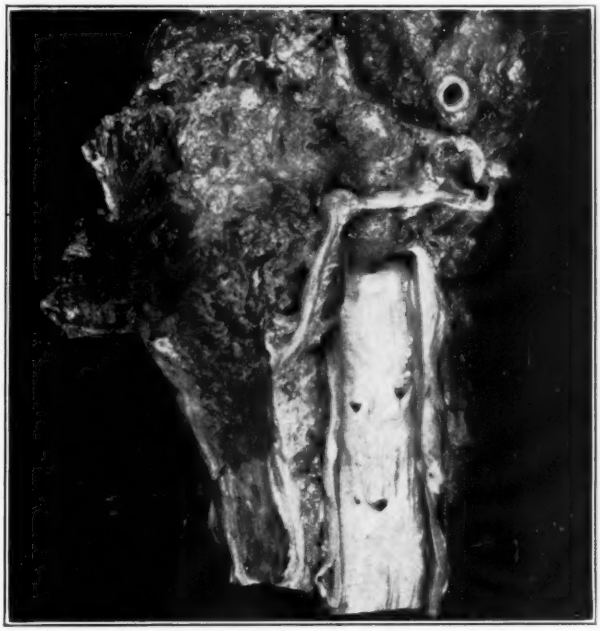

Fig. 1.—Tumor thrombus (Case 1) extending from right renal vein into inferior vena cava and left renal vein; tumor thrombosis extends upward, induced thrombosis extends downward from a distance 2 cm. below entrance of right renal vein. The aorta is opened below the left renal vein.

94 per cent; there were 4,020,000 erythrocytes and 7,000 leucocytes in each cubic millimeter of blood. A roentgenogram of the thorax did not disclose any abnormality. The value for the urea was 44 mg. per 100 c.c. of blood. Urinalysis revealed a few erythrocytes. The bromsulphalein test disclosed moderate impairment of liver function. The electrocardiogram disclosed right ventricular preponderance.

Two days after the patient came to the clinic, abdominal paracentesis was performed and 2,100 c.c. of fluid was removed. Four days later the patient went into a state of shock and died.

Necropsy disclosed slight edema of the ankles. The peritoneal cavity contained 25 c.c. of ascitic fluid. The right kidney weighed 745 gm. Its lower pole had been replaced completely by soft yellowish tumor tissue. The surface of the kidney was coarsely nodular. In several places cauliflower-like masses of tumor

tissue had perforated the renal capsule. The tumor measured 12 by 6 by 7 cm. and was very friable. Its cut surface was yellowish-red. The upper half of the kidney contained metastatic nodules; approximately 95 per cent of the substance of the kidney had been replaced by the tumor. The tumor extended into the right and left renal veins and the inferior vena cava. Friable tumor tissue extended downward in the inferior vena cava for a distance of 2 cm. (Fig. 1). Below this tumorous thrombosis was a simple thrombosis which extended downward into both iliac veins. The latter had a homogeneous or laminated appearance and was grayish red. The tumorous thrombosis extended upward beyond the hepatic veins; it extended into the right auricle and through the tricuspid valve and into the right ventricle. It was firmly attached throughout its extension in the inferior vena cava. In the right auricle and right ventricle the tumorous thrombosis was loosely adherent and was attached only to the anterior wall of these chambers. The pulmonary valve was partly occluded by the tumorous thrombosis, but there was no evidence of pulmonary embolism.

The liver weighed 1,820 gm. and was reddish brown. The consistency of the right lobe was increased slightly. Metastatic nodules were present in the right and left lobes of the liver. There was diffuse chronic passive congestion of the liver; this was particularly marked in the right lobe. The collateral circulation was not marked in the body; this possibly might be attributable to the fact that the body had been embalmed before the necropsy was performed. There was evidence of venous engorgement throughout the small intestine.

Histologic examination disclosed that the tumor was a papillary carcinoma (hypernephroma); however, it contained fewer clear cells than usually are found in this type of tumor.

Case 2.—A white woman, aged 33 years, first came to the clinic Aug. 23, 1936, because of intermittent attacks of epigastric distress which had been relieved by food or alkalies. A roentgenogram of the stomach and duodenum revealed a duodenal ulcer, and the patient was instructed in a medical regimen for the ulcer. The patient came to the clinic again on Nov. 23, 1936, because of irregular and excessive menstruation. A dilatation and curettage revealed an atrophic cystic menopausal type of endometrium. At the time of this visit to the clinic the patient complained of swelling of the left leg. The swelling of the leg had been present for only a few days. The edema of the left leg persisted and in the first part of February, 1937, edema also developed in the right leg.

The patient was seen again on Feb. 24, 1937, and a diagnosis of acute perforation of a duodenal ulcer was made. Operation was performed immediately and the perforation was closed. At this time (Feb. 24, 1937), it was noted that the edema of both legs had increased.

Examination on Feb. 24, 1937, revealed a pale and obviously very ill woman who was having severe pain. Examination of the abdomen revealed rigidity and excruciating tenderness over the entire abdomen. There was a severe edema present over both legs and over the sacrum. The edema was greatest over the left leg. Examination of the heart and lungs was essentially negative except for the rapid pulse rate. The values for the blood pressure were 100 mm. of mercury systolic and 68 mm. diastolic. During the patient's stay in the hospital from Feb. 24, 1937, to April 21, 1937, the concentration of hemoglobin ranged from 79 to 89 per cent; the erythrocyte count ranged from 3,820,000 to 4,380,000 per cubic millimeter, and the leucocyte count ranged from 9,200 to 16,600 per cubic millimeter of blood. A roentgenogram of the thorax did not disclose any abnormality. The flocculation tests for syphilis were negative. The value for the urea ranged from 20 to 40 mg. per 100 c.c. of blood. Fifteen urinalyses revealed erythrocytes in the urine on only one occasion, and on that occasion only an occasional erythrocyte was found in the urine. There were pus cells in the urine on several occasions.

On Feb. 28, 1937, four days after the closure of the perforated duodenal ulcer, jaundice developed; this persisted for several days. On March 2, 1937, six days after the duodenal operation, a severe ascites developed. This necessitated a reopening of the incision, which drained serous fluid for several days. The patient had a parotitis on the left side on March 6, 1937; this subsided after treatment with radium. On March 16, 1937, the patient began having large, foamy, yellow stools which were thought to be due to a pancreatitis; this intestinal condition cleared up spontaneously within a few days. Throughout her entire postoperative course the patient's pulse rate ranged from 100 to 140 although her temperature was never more than 100° F.; the patient had continued to have persistent edema of the legs and the sacrum and a marked ascites with dilatation of the superficial abdominal veins. The ascites and edema were partially controlled by frequent injections of

Fig. 2.—Cut surface of carcinoma of right kidney (Case 2) showing extension of tumor thrombus into renal vein and upward into inferior vena cava. Tags of liver tissue are attached to the inferior vena cava at the top of the specimen.

salyrgan and restriction of the fluid intake. The patient was dismissed from the hospital on April 21, 1937. The patient was seen at frequent intervals at her home; the injections of salyrgan were continued and the fluid intake was restricted. Despite these measures the ascites increased; on May 8, 1937, a paracentesis was performed and 1,200 c.c. of clear bile-colored fluid was removed. Following this, the patient continued with very little change until June 24, 1937, when chills and fever developed; the patient became cyanotic and died on June 28, 1937.

Necropsy disclosed a definite edema of the feet and legs. The skin was slightly icteric. There was a ventral hernia in the upper part of the abdomen in which the hernial sac was distended to 23 by 21 cm. in diameter. The peritoneal cavity contained 7 liters of clear yellow fluid. The right kidney weighed 500 gm. The upper

two-thirds of this organ was occupied by a fibrous encapsulated yellowish-red tumor which measured 8 by 6 by 6 cm. and communicated directly as a tumor thrombus with the renal vein (Fig. 2). This vein measured 7 cm. in diameter. Approximately 50 per cent of the substance of the right kidney had been replaced by the tumor. The left vein was not thrombosed. The tumor thrombus extended directly from the right renal vein into the inferior vena cava which was distended

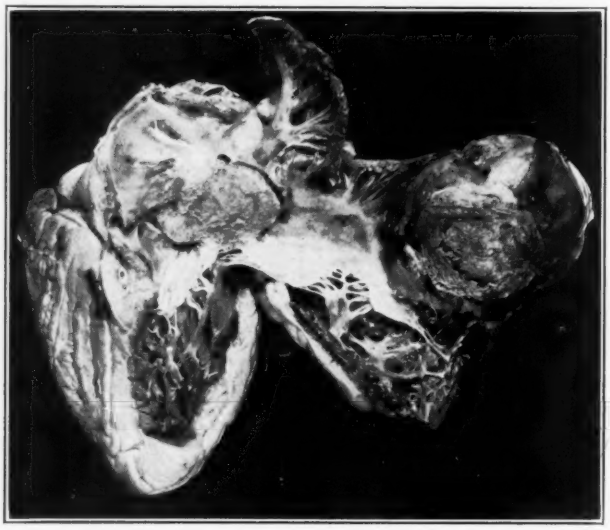

Fig. 3.—Tumor thrombus in left auricle (Case 2); continuity with the tumor thrombus in the inferior vena cava was severed along the rough round area on the lower surface of the mass. The tumor also was attached to the roughened surface of the interauricular septum.

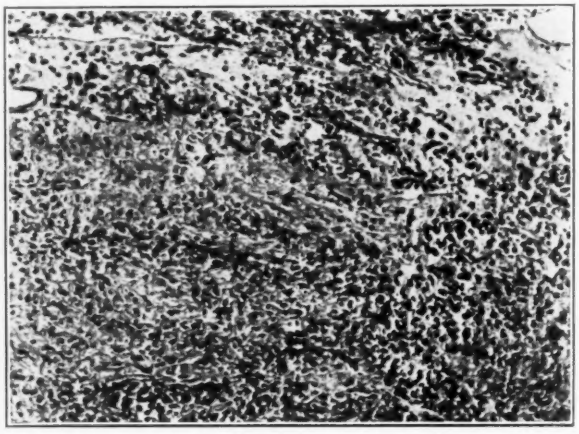

Fig. 4.—Microscopic appearance of the tumor in Case 2; one may note the general resemblance to sarcoma; the cells to left of the center closely resemble clear epithelial cells of renal carcinoma; specimen stained with hematoxylin and eosin (×95).

to a diameter of 9 cm. (Fig. 3). The tumorous thrombosis extended upward into the right auricle where it formed a mass 6 by 5 by 4 cm. This mass was firmly attached to the auricular endocardium, and obstructed the opening of the tricuspid valve at least 95 per cent. There was no pulmonary embolism.

The liver weighed 2,100 gm. and was dark red. Severe chronic passive congestion was present. The hepatic veins were not thrombosed. There were marked varicos-

ities in the lower part of the esophagus. The stomach contained 50 c.c. of dark, red, partially digested blood. Both right and left femoral and iliac veins were thrombosed. There were scars on the anterior and posterior walls of the first portion of the duodenum, and a contracted line of scar tissue joined the two scars. The anterior scar appeared on the serosa and was apparently the point of the former perforation. There was no active ulcer.

Histologic examination disclosed that the tumor was pleomorphic and vascular. It contained many regions of necrosis and hemorrhage (Fig. 4). The cells were of various sizes and shapes and contained hyperchromatic nuclei. There was no well marked stroma or pattern of arrangement of the cells of the tumor. In some regions there was a honeycomb appearance of a group of contiguous cells in which the cytoplasm was clear. This appearance resembled that of vegetable fibers and was the only characteristic of renal carcinoma found microscopically. It is possible that an unequivocal histologic diagnosis of carcinoma is subject to question and that some pathologists might call this tumor a sarcoma. It is our opinion, however, that, since renal tumors are notoriously pleomorphic in microscopic appearance, we can accept this tumor as a carcinoma (hypernephroma) with poor histologic differentiation.

CASE 3.—A white man, aged 62 years, came to the clinic because of recurrent attacks of right renal colic and associated dysuria. The first attack had occurred seven years before the patient came to the clinic. On one occasion hematuria had occurred. Examination at the clinic disclosed a palpable, indefinite, fixed mass in the right renal region. Urinalysis revealed a few erythrocytes and a few pus cells. A pyelogram revealed a tumor of the right kidney. Operation disclosed a large adherent renal tumor that extended into the right renal vein. The patient died fourteen days postoperatively. Necropsy disclosed metastatic involvement of the liver, lungs, and adrenal glands. The carcinoma extended into the right renal vein and the inferior vena cava to the level of the diaphragm.

CASE 4.—A white man, aged 46 years, came to the clinic because of intermittent hematuria. The hematuria had been first noted five years before the patient came to the clinic. The patient had had urinary frequency and dysuria and a mild pain in the back, which had been more severe on the right side and had been associated with the attacks of hematuria. Roentgenographic examination had disclosed a tumor of the right kidney two years before the patient came to the clinic. Examination at the clinic disclosed a moderately enlarged right kidney. Urinalysis revealed occasional erythrocytes and a few pus cells. A pyelogram revealed a tumor of the right kidney. A large renal tumor, which extended beyond the midline and was fixed to the spinal column, aorta and vena cava was found at operation. Pneumonia developed and the patient died fifteen days after the operation. Necropsy disclosed that the carcinoma had extended into the right renal vein and the inferior vena cava. There was metastatic involvement of the liver, lungs, and the adrenal glands on the right side.

CASE 5.—A white man, aged 72 years, came to the clinic because of epigastric pain, constipation, and continued loss of weight (37 pounds). These symptoms first had been noted three months before the patient came to the clinic. A day after the patient came to the clinic, severe generalized abdominal pains and persistent vomiting developed. The patient was treated conservatively. There was no history of any urinary symptoms. The results of examination at the clinic were essentially negative except for slightly dilated varicosities of the lower extremities. Urinalysis revealed numerous erythrocytes and an occasional pus cell. Because of the persistent intestinal obstruction, an ileostomy was performed five days after the patient came to the clinic. The patient continued to fail and died

thirty-five days after the operation. The patient's condition was too critical to warrant any further urologic investigative procedures. Necropsy disclosed a carcinoma (hypernephroma) of the right kidney which had extended into the inferior vena cava. There was metastatic involvement of the liver and lungs. The intestinal obstruction was not related to the renal carcinoma.

CASE 6.—A white man, aged 60 years, came to the clinic because of a sensation of fullness over the right side of the abdomen and recent slight loss of weight. The sensation of fullness over the right side of the abdomen first had been noted four years before the patient came to the clinic. Examination at the clinic disclosed a large, fixed right kidney. Urinalysis revealed a few erythrocytes and a few pus cells. Excretory urograms and a right pyelogram revealed a tumor of the right kidney. Operation disclosed a large renal tumor which was adherent to the surrounding structures and had extended into the inferior vena cava and to the retroperitoneal portion of the duodenum. The patient died fifteen days after the operation. Necropsy disclosed that the carcinoma (hypernephroma) had extended into the right renal vein and the inferior vena cava, 3 cm. above the renal vein. There was tumorous embolism of the lungs.

CASE 7.—In this case the patient never was examined at the clinic. Necropsy only was done. The patient was a white woman, aged 71 years, who had died suddenly. Her relatives said the patient had had hematuria for two months and that she had had an enlarging mass in the left jaw for three weeks before she died. No other data were available. Necropsy disclosed a carcinoma (hypernephroma) of the left kidney. The tumor had extended into the right renal vein and the inferior vena cava. There was metastatic involvement of the lungs.

COMMENT

The findings in these 7 cases are in accord with the findings of the 34 cases that have been reported in the literature. In 5 of the 7 cases the patients were men, and in 28 of the 34 cases previously reported the patients also were men. In 6 of the 7 cases the tumor was on the right side and it appeared on the right side in 24 of the cases previously reported. Edema or ascites was present in only 2 of our cases, while edema or ascites occurred in 24 of the cases previously reported. Pain of varying degree, character, and situation was present in 4 of our cases and in 21 of the cases previously reported. Engorgement of the collateral superficial veins was found in 2 of our cases and in 11 of the cases previously reported. In only 3 of our cases was there a history of gross hematuria. In 3 other cases an occasional erythrocyte was found in the urine.

Extension of the tumorous thrombosis into the right auricle was reported in 16 of the 34 cases reported previously. This occurred in 2 of our cases. In one case (Case 1) the tumorous thrombosis extended into the right auricle and through the tricuspid valve into the right ventricle, and in the other case (Case 2) it extended into the right auricle. This was a most interesting finding and was especially striking because of the paucity of symptoms. Neither of these patients had had very severe dyspnea. The results of examination of the heart

were essentially negative except in Case 2, in which the patient had a persistently high pulse rate. The lack of cardiac symptoms is similar to the lack of symptoms found in chronic constricting pericarditis. The similarity is further shown by the fact that they both give rise to the same mechanical embarrassment of the heart, namely, an inflow stasis.

Thrombosis of the hepatic vein was present in one case (Case 1). In this case metastatic nodules also were present in both right and left lobes of the liver; the liver was enlarged and the bromsulphalein test revealed moderate impairment of liver function. There was no history of jaundice. In another case (Case 2) the jaundice developed four days after operation for a perforated duodenal ulcer. The jaundice was present for several days but cleared up spontaneously. In this case, the hepatic veins were not thrombosed, and there was no evidence of metastatic involvement of the liver; the only finding was a severe chronic passive congestion of the liver. We are unable to explain the presence of jaundice in this case. However, jaundice of unknown origin is not infrequently seen in severe intra-abdominal accidents, such as perforated duodenal ulcer, and the jaundice in this case may have been on the same basis.

The most striking feature in the 7 cases reported is the lack of clinical symptoms. In the first 2 cases (Cases 1 and 2) the diagnosis of thrombosis of the inferior vena cava was suggested, but was not made definitely until necropsy. In one case (Case 2) there were no symptoms that were suggestive of a renal affair at any time and it is significant that a renal tumor can be so extensive and produce no renal symptoms. In another case (Case 5) the renal tumor with extension to the inferior vena cava was an incidental finding and did not directly contribute to the death of the patient.

The classic signs of obstruction of the inferior vena cava, namely, engorgement of the superficial veins, edema, and ascites, were present in only 2 cases. In the remaining 5 cases these signs were not present. One, therefore, must conclude that with our present diagnostic facilities and knowledge it is very difficult to make a clinical diagnosis of obstruction of the inferior vena cava and in most instances the diagnosis must be made at necropsy.

SUMMARY

Edema, ascites, and engorgement of the collateral superficial circulation, which would indicate the presence of obstruction of the inferior vena cava, were present in only 2 of the 7 cases. Extension of the tumor thrombus into the right auricle occurred in 2 cases. Thrombosis of the hepatic veins and metastatic involvement of the liver with impaired liver function occurred in 1 case.

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THE ROLE OF UREA-SPLITTING ORGANISMS IN THE FORMATION OF CERTAIN TYPES OF STONES IN THE URINARY TRACT

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THE relationship of alkaline infections of the urine to the formation of certain types of stone in the urinary tract is a problem that has been of interest to investigators for many years. In 1897 Horton Smith[1] described a new variety of the proteus group, *Bacillus proteus urinae*, discovered in the urine of a patient suffering from cystitis. He pointed out that the strain he was investigating decomposed the urine, setting free ammonia and precipitating triple phosphates. In 1901 Thomas Brown,[2] of Baltimore, stressed the influence of certain kinds of organisms upon the composition of stones in calculous pyelonephritis. Of the six cases he presented, five had an alkaline urine, three infected with *Bacillus proteus* and two with white staphylococci. All five stones were alike chemically, being composed chiefly of the phosphates and carbonates of calcium and magnesium. In 1915 Wallace and Dudgeon,[3] of St. Thomas Hospital in London, reported the case of a man, aged 60 years, who developed an infection of the urinary tract and septicemia, following suprapubic prostatectomy. The *Bacillus proteus* was cultured from both the blood stream and urine. They described the cultural characteristics of the organism but did not mention the relationship of this type of infection to possible stone formation. The case is referred to in order to emphasize isolated efforts to study urinary bacteriology more than twenty years ago.

In 1925 Shaw and Hill[4] described another new organism, a small gram-positive bacillus, which they named the *Corynebacterium thompsoni*. It splits urea at a rapid rate and infection only occurs in wounds that are the site of urinary drainage. This organism is the most virulent of the urea-splitting group, but fortunately one that is not commonly found.

In addition to proteus and Thompson's bacilli, other organisms that have been found to decompose urea and produce an alkaline urine are certain strains of staphylococci, streptococci, pyocyaneus, occasionally some strains of *B. coli*, and rarely some of the diphtheroids. *Bacillus proteus* is the organism usually found in urea-splitting infections. Out of 31 cases studied by the late Dr. Joseph Hume over a period of four years, it was present 26 times. In the other 5, staphylococci were found

Received for publication, June 22, 1938.

in 2, diphtheroids in 2, and paratyphoid in 1. The importance of early recognition of this group of organisms is generally appreciated. They lead to early stone formation and produce a rapid destruction of renal tissue.

So far as the association of infection with stones is concerned, Joly[5] in 1929 made his viewpoint quite clear. He stated that, if the urine is

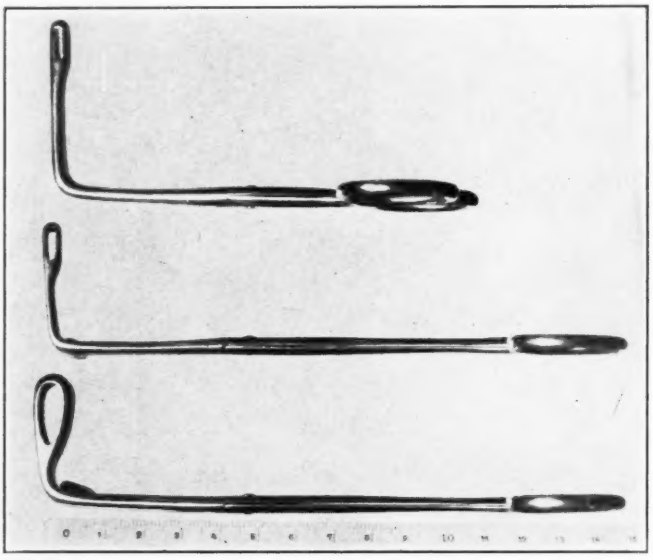

Fig. 1.—Hume stone forceps, three sizes.

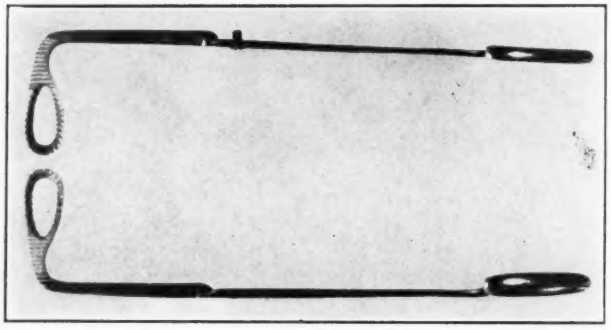

Fig. 2.—Hume stone forceps opened showing beveled corrugated edge.

acid, the stone will behave as if the urine were not infected; that is, it will grow slowly and will not as a rule have a covering of phosphates. The infection, however, will persist until after the stone is removed. On the other hand, an alkaline infection is more serious. The stone will become coated with phosphates, will grow rapidly, and will show a marked tendency to recur after removal.

Armed with all the facts presented up to that time and stimulated by an increasing number of recurrent stones, Hume, early in 1930, set about

to formulate a more satisfactory plan of handling such cases. After a detailed study of 31 cases over a period of four years, he settled upon a plan which in the main involved three principles: (1) the preliminary study, with particular attention directed toward the type of infection, (2) operative treatment with special reference to drainage, and (3) a particular method of postoperative management.

In the preliminary study an immediate attempt was made to separate infected cases into (1) those associated with an acid urine and (2) those in which the organisms present decomposed urea with the production of an alkaline urine. The usual routine procedures shall be taken for granted. The reaction of the urine as determined by a pH indicator suggests the group into which the case may fall. Smears are made of the sediment from a centrifuged specimen and stained by Gram's method.

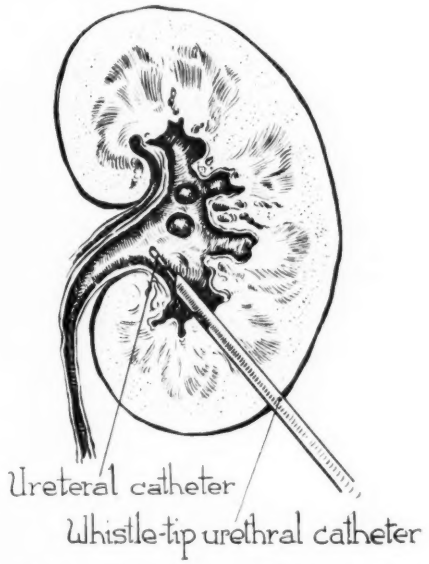

Fig. 3.—Nephrostomy tube in place after ureteral catheter has been inserted for continuous drip.

From this one determines whether the infection is coccal, bacillary, or mixed, and whether the organisms are gram-positive or gram-negative. The next step is to determine whether or not the organisms present possess the property of decomposing urea. To settle this question, Hume inoculated a tube of veal broth containing 2 per cent urea with 0.2 per cent phenolphthalein added as an indicator. The tube is placed in an incubator, kept at 37° C., and examined at intervals for the next twenty-four hours. If the organisms present decompose urea, ammonia is produced, creating an alkaline reaction and consequently a red color, due to the presence of phenolphthalein in the medium. On the other hand if the color remains amber, the organisms do not split urea. This method is simple and inexpensive in that it can be carried out in a small in-

cubator. It enables one within twenty-four hours of the time the case is first seen to label it as being of the urea-splitting or nonurea-splitting variety. For practical purposes, this may be all that is necessary. It has been our custom, however, to identify the particular organisms present by detailed cultures in the hospital laboratory. A complete diagnosis

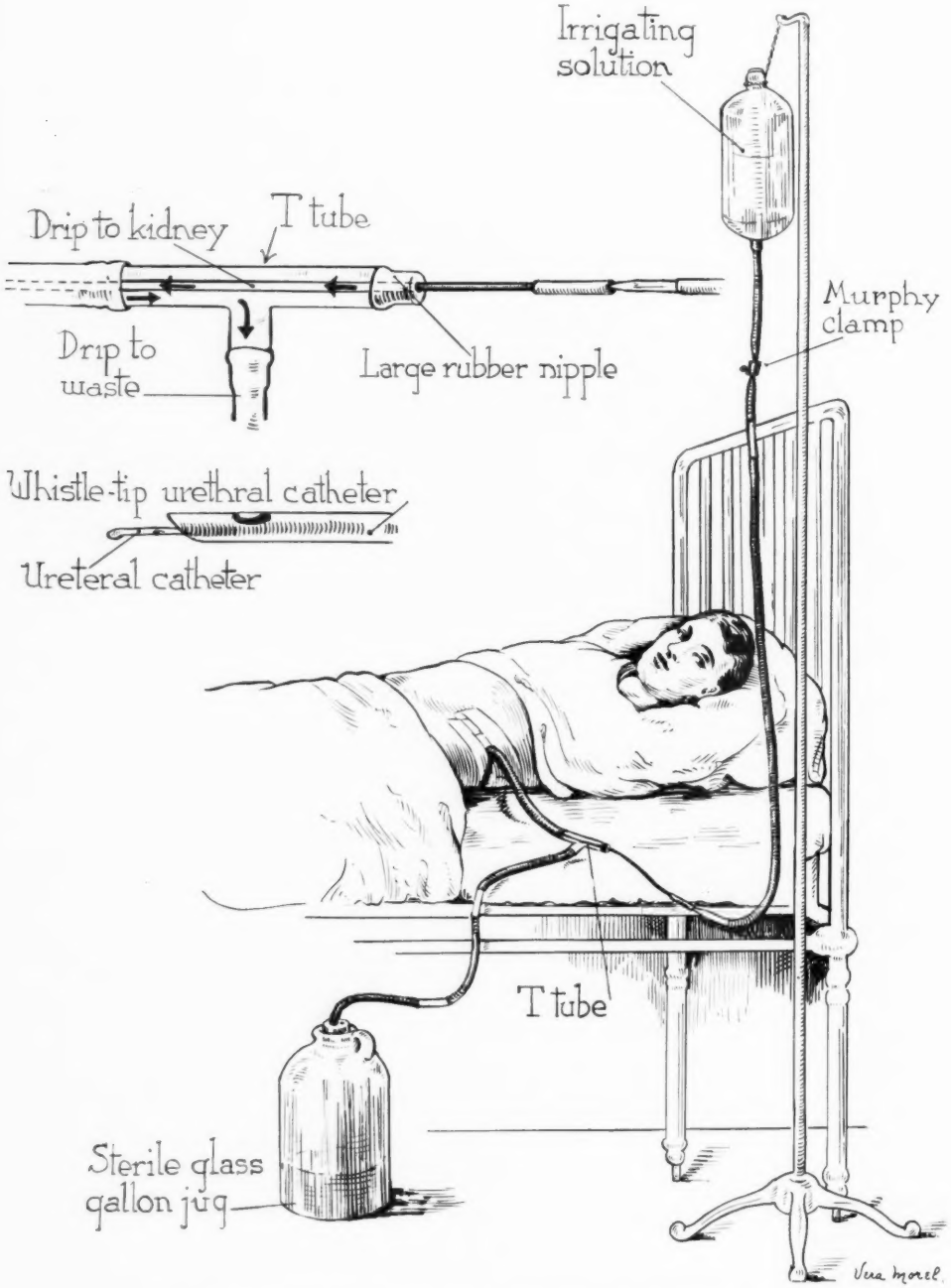

Fig. 4.—Complete set-up for giving continuous irrigation.

of stone implies that the usual preliminary studies have been done, and, when one adds to that a detailed knowledge of the associated infection, the indications in treatment become quite clear.

So far as operative treatment is concerned, the aim of a well-planned attack is to remove the stone, preserve kidney tissue, combat infection, and prevent recurrence. Such operations, on the whole, demand wide exposure, gentle handling, certainty of removal of all fragments and drainage via the cortex, through the lower calyx into the pelvis. This involves chiefly the application of principles long recognized. In order to facilitate the removal of stone fragments and protect the kidney from trauma, Hume devised special stone forceps shown in Figs. 1 and 2. We have found these most useful in handling both single and multiple stones. We were able on one occasion to remove 206 small stones from

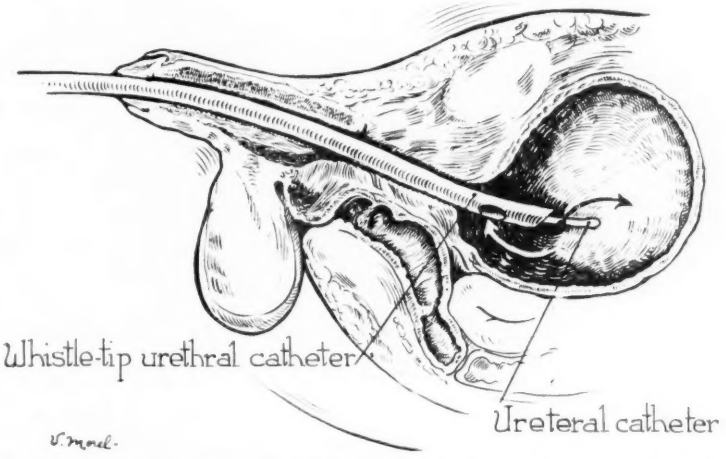

Fig. 5.—Arrangement for continuous bladder irrigation (Foley catheter may be substituted in bladder cases).

a kidney through a pelvic incision, leaving but a single fragment, which was later washed out through the nephrostomy tube. Drainage by nephrostomy tube was regarded as an essential in every case where the kidney was found to be infected with any of the urea-splitting group of organisms. It was carried out regardless of the size and number of stones removed. In addition to the beneficial action of drainage on the inflammatory lesion, it provides an opening through which small fragments may be removed and an easy means to combat infection with intermittent or continuous irrigation.

After removal of the stone, whether or not the patient is faced with recurrence is almost entirely dependent upon the efficacy of the treatment used to clear up the infection. Randall[6] in 1932 advocated the use of phosphoric acid irrigations for this purpose. Strengths of 1 per cent, occasionally 3 per cent, were used, and the results obtained in

eleven cases reported. Intermittent irrigations were given and the solution was apparently nonirritating to the urinary tract. Acting upon this suggestion, Hume in 1934 began to employ continuous irrigations by means of a two-way catheter arrangement, shown in Figs. 3, 4, and 5. He tried various solutions but agreed with Randall that by the use of

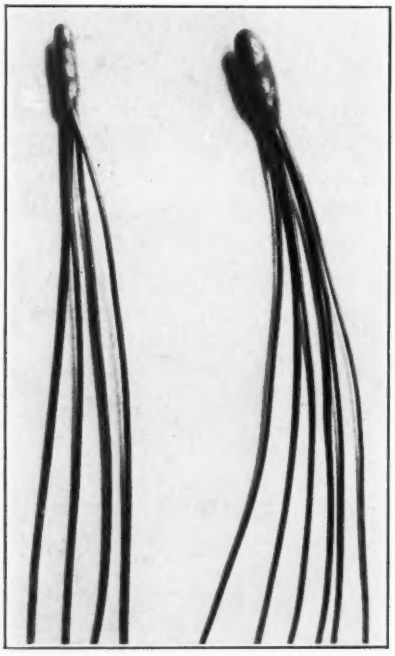

Fig. 6.—Fused ureteral catheter for removing stone fragments from renal pelvis.

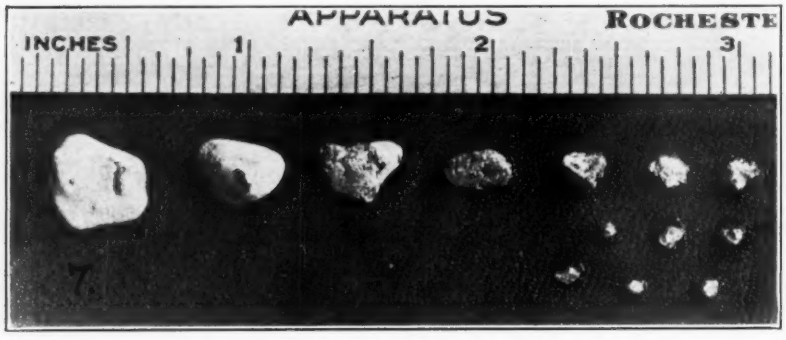

Fig. 7.—Stone fragments from a single kidney removed by catheters shown in Fig. 6.

phosphoric acid the best results were obtained. For continuous irrigation, ½ per cent solution was used in the majority of cases. Occasionally, however, a 1 per cent strength may be tolerated. The rate of flow is controlled by a Murphy clamp placed on the tube below the infusion bottle as shown in Fig. 4. The solution is allowed to flow at a rate of 20 to 40 drops per minute, according to the tolerance of the

individual case. If there is pain or a feeling of fullness in the kidney, the flow should be cut down. Continuous irrigation may be started by the end of the first postoperative week and continued for two or three weeks, by which time intermittent irrigations usually suffice. The intermittent irrigations are continued until the cultures are negative for urea-splitting organisms. After the patient is able to be up and about, he is taught to handle his tube and give his own irrigations.

Fragments left at operation or formed during the postoperative period present an unpleasant problem. It is impossible to clear up the infection as long as they are present, and, if allowed to remain, they soon grow to a size too large to be removed through the nephrostomy opening. Confronted with this type of problem in a case with a single kidney, and having failed to catch the fragment with various types of forceps, Hume fused the ends of No. 4 French ureteral catheters together (Fig. 6) and removed thirteen stone fragments (Fig. 7) from the pelvis of the kidney. The tips of the catheters are fused with dental wax and the bulb is made only large enough to hold them together with a firm grip. The nephrostomy tube is removed, the bulb passed into the renal pelvis, and each catheter pushed in separately for a considerable distance. After being coiled up in the pelvis, the stone fragment is almost invariably caught behind the wax bulb as the catheters are straightened out again to be removed. We have repeatedly removed fragments from the renal pelvis in this manner but have yet to catch one confined to either of the calices.

SUMMARY

An attempt is made to emphasize the relation of alkaline infections to stone formation in the urinary tract. Useful methods worked out by the late Dr. Joseph Hume are presented.

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THE USE OF VITAMIN B_1 IN THE PREOPERATIVE PREPARATION OF THE HYPERTHYROID PATIENT

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IN NO group of surgical patients is the problem of nutrition more urgent than in the hyperthyroid patient. It has long been known that the problem is not entirely one of providing an adequate caloric intake, for even though the caloric intake, in certain instances, has been satisfactory, maintenance of weight is often not obtained. It is probable that certain accessory food factors may be deficient during the hyperthyroid state and that the deficiency may influence the clinical picture and the course of the disease. Weiss and Wilkins[1] have described the cardiac effects of a B_1 deficiency.

We have observed patients whose response to iodine therapy was highly satisfactory from the viewpoint of a reduction in the basal metabolic rate and even the pulse rate, but who continued to have anorexia and to exhibit weight loss. Other patients have maintained a tachycardia in spite of a reduction in the basal metabolic rate, while still others have lost weight out of proportion to the increase in the basal metabolic rate and the appetite level. These observations suggest that, although the food intake in total calories may be sufficient, absorption or utilization of the ingested foodstuffs may be below the normal.

That one of these factors may be the vitamin B complex, or one of its components, is suggested by the close relationship between this vitamin and thyroid function. Cowgill and his co-workers[2] and Hendricks[3] have shown that the vitamin B complex, and especially the B_1 requirement in animals and man, bear a direct relationship to metabolism. It is said that this relationship can be expressed in a mathematical formula.[2] If the metabolic rate is raised by disease or the administration of thyroxin, the vitamin B_1 requirement is proportionately increased and additional B_1 must be added to the diet to prevent the appearance of the symptoms of a B_1 deficiency. That this deficiency may develop rapidly is suggested by the studies of Brodie and MacLeod,[4] which indicate a very limited capacity for storage of vitamin B_1 in human tissues. Harris and Leong,[5] from studies of the excretion of B_1 in the urine, concluded that the amount of this vitamin stored in the body was quite small and that the main-

Received for publication, June 10, 1938.

tenance of the vitamin B_1 stores was dependent on an adequate intake in the diet. They believe that it is possible to tell from the amount of B_1 daily excreted in the urine whether the intake of this vitamin is adequate.

There is suggestive evidence that a need for increased amounts of B_1 exists in the patient with hyperthyroidism. That this need is not satisfied in many patients with hyperthyroidism is very probable, especially if, as Sherman[6] and Sure[7] have stated, the average American diet is becoming more and more deficient in this vitamin. There is, moreover, clinical evidence that such a deficiency exists in many instances. Gastrointestinal symptoms are among those most commonly associated with simple B avitaminosis (Buchanan,[8] Elsom[9]). Of these, anorexia is the most frequently encountered; a feeling of fullness in the epigastrium, gaseous eructations, constipation followed by, or alternating with, diarrhea are often associated with the anorexia. Hypo- or achlorhydria and changes in the motor function of the gastrointestinal tract are also reported by many observers in patients deficient only in the B_1 fraction (Cowgill and Gilman[10] and Fitts[11]).

Similarly, although most hyperthyroid patients have increased appetite at the onset of the disease, the majority develop anorexia as the thyrotoxic symptoms are maintained. Hinwich, Goldfarb, and Cowgill[12] have shown that anorexia develops much more rapidly in dogs fed thyroxin and a B_1 deficient diet than in those getting an adequate diet plus the same amount of thyroxin. These authors suggested, on the basis of their findings, that a B_1 deficiency may be the important factor in the anorexia and weight loss of hyperthyroid patients.

The other gastrointestinal findings previously mentioned as evidence of a B_1 deficiency are also frequently present in thyrotoxic patients. Complete achlorhydria, even after histamine, has been found in 35 per cent of a series of patients with hyperthyroidism recently studied at this hospital.[13] Delayed gastric emptying time, a common finding in patients with B_1 avitaminosis, has also been found in many of the above-mentioned patients.

There is, therefore, evidence that a B complex, or B_1, deficiency may play a part in the production of certain of the gastrointestinal disturbances, especially the anorexia often exhibited by the hyperthyroid patient.

Considerable literature has accumulated dealing with the effect of diets deficient in vitamin B_1 or the B complex on the cardiovascular apparatus of animals and man (Weiss and Wilkins,[1] Sankaran and Krishnan,[14] Jones and Sure,[15] and Zoll and Weiss[16]). Although in some species of animals bradycardia is frequently encountered, the findings most common in man are tachycardia, palpitation, dyspnea, fatigability, and a lowered exercise tolerance. Certainly no more

characteristic list of the major cardiovascular symptoms associated with hyperthyroidism could be compiled. If these observations are correct, and we believe they are, they suggest that a vitamin B deficiency may intensify the changes in the cardiovascular mechanism produced by thyrotoxicosis.

The possibility that a lack of the vitamin B complex may be the initiating factor in the development of hyperthyroidism has been investigated. As early as 1914, McCarrison[17] noticed hyperplasia of the thyroid gland of pigeons fed on B deficient diets. Similar findings in other experimental animals have been reported by Stepp and György,[18] Spence,[19] and Fischer.[20] Sandberg and Holly[21] have shown that the addition of vitamin B_1 in adequate amounts to the diet of such animals will produce involution of the hyperplastic gland. Although Carpenter and Sharpless[22] were unable to corroborate their findings, the possibility that this same train of events may occur in humans is suggested by a recent report in which Means, Hertz, and Lerman[23] suggest that certain cases of Graves' disease may be caused by severe degrees of malnutrition due to hopelessly inadequate diets. Certainly a vitamin B_1 deficiency must have developed rapidly in these patients, before the symptoms of thyrotoxicosis appeared.

In an attempt to put the antithyrogenic action of vitamin B_1 to a therapeutic test, Sure and his associates[24] studied the effect of vitamin B_1 administration upon experimentally produced hyperthyroidism. They found that by means of a highly concentrated vitamin B_1 extract they were able to reduce the injurious effect of thyroxin in the rat. Later they were abe to protect these animals entirely from the toxic influences of thyroxin by the parenteral administration of crystalline vitamin B_1. The dosage of vitamin required was very large. Recently these authors[25] reported that, although vitamin B_1 has some antithyrogenic action, it is more effective if the other components of the B complex are added.

The role of vitamin B_1 in carbohydrate metabolism offers a clue as to the mechanism by which this substance may counteract in part the effects of thyroxin. That one of the most important functions of B_1 is the part it plays in the proper utilization of carbohydrates has been shown by Plimmer and Rosedale,[26] Peters,[27] Drill,[28] and Abderhalden and Wertheimer.[29] They[26] demonstrated that the administration of additional carbohydrate to the diet of various animals markedly increased their requirement for the B_1 fraction. If this requirement is not met, hyperglycemia and depletion of liver and muscle glycogen result.[30-32] If vitamin B_1 is then supplied in adequate amounts, the blood sugar falls and glycogen storage in the liver is increased.[33]

It is well known that thyrotoxicosis is commonly associated with disturbances in carbohydrate metabolism. These changes are char-

acterized by hyperglycemia, decreased glucose tolerance, and depletion of liver glycogen. The similarity between these changes and those attributed to B_1 deficiency is noteworthy.

Clinicians are now agreed that one of the most important steps in the treatment of severe grades of thyrotoxicosis is to provide an abundant supply of carbohydrates in a readily assimilable form, either orally or parenterally, so that the patient may rapidly replenish his glycogen stores (Frazier and Brown,[34] and Lahey[35]). The value of this form of therapy, both before and after operation, has been thoroughly established. If thyrotoxic patients are deficient in vitamin B_1, which in itself aids in the deposition of glycogen, it is equally important to supply this factor during the period that the carbohydrate intake is being increased.

From the evidence presented, it is reasonable to believe that some degree of vitamin B_1 deficiency must exist in patients with moderate or severe hyperthyroidism. A comparison of the symptomatology of the two conditions reveals certain striking similarities. That B_1 avitaminosis may contribute at least to an intensification of the thyrotoxic symptoms is a justifiable assumption. Although vitamin B_1 probably has no direct antithyrogenic action in man, the administration of adequate amounts of this vitamin should be an important factor in the treatment of severe clinical hyperthyroidism.

EXPERIMENTAL OBSERVATIONS

Several years ago we began the use of a B complex extract in patients suffering from hyperthyroidism admitted to the Hospital of the University of Pennsylvania. The patients were given large amounts of the extract with no other therapy during the period of observation. It was our impression that, although the basal metabolic rate was not reduced any more than in a control group, the general condition of the vitamin-treated patients was better than the control group. In order to evaluate the beneficial effects, if any, which were derived from the use of the vitamin, the progress of a second group of patients was compared with that of a group similarly treated in every respect except that no vitamin B was administered. The control group was composed of twenty-eight patients. The routine preoperative treatment was complete bed rest except for two hours daily; high caloric, high carbohydrate diet, 3,000 to 4,000 calories per day; saturated solution of potassium iodide, minims 5 to 10 three times a day; sodium bromide, 10 gr. three times a day, and phenobarbital, 1½ gr. nightly.

The basal metabolic rate was determined on the day following admission and at three- or four-day intervals thereafter. The patients were all weighed daily. Fifty patients composed the vitamin-treated group. In addition to the above outlined regime, these patients re-

ceived hypodermically 10 mg. of crystalline vitamin B_1* every other day, and 10 gm. of Brewers' yeast daily by mouth.

The yeast tablets administered to the first half of this group provided a daily intake of approximately 1.5 mg. of B_1 and the remainder received approximately 1.0 mg. of vitamin B_1. This means that all of the patients received between 6 and 7 mg. of vitamin B_1 daily, which is about two and one-half times their daily requirement as calculated from Cowgill's[2] formula.

TABLE I

	NUMBER OF CASES	PREOPERATIVE PERIOD		POSTOPERATIVE REACTION	
		PULSE DROP	B.M.R. DROP	PULSE RISE	TEMPERATURE
Control					
A	14	19	39	34	102.2
B	8	10	20	29	101.6
C	6	6	8	22	101.0
Total	28				
With B_1+					
A	28	26	38	38	101.8
B	13	16	14	33	101.6
C	9	8	15	38	101.0
Total	50				

Each series was divided into three groups, Group A being those whose basal metabolic rate on admission was over 40; Group B, between 30 and 40; and Group C, those between 20 and 30. The various criteria of comparison are outlined in Tables I and II. It will be seen that only those patients whose admission metabolic rates are within the same range are compared (Tables I and II).

TABLE II

	NUMBER OF CASES	PREOPERATIVE HOSPITAL DAYS	AVERAGE WEIGHT CHANGE (POUNDS)	GAINED WEIGHT (POUNDS)	LOST WEIGHT (POUNDS)	NO CHANGE	APPETITE	
							GOOD	FAIR TO POOR
Control								
A	14	15	−1.2				16 cases	12 cases
B	8	9.5	−0.6				31.2% gained weight	25% gained weight
C	6	7	0.0					25% improved
Total	28	11.1	−0.4	2.8 (28.5%)	3.0 (46.5%)	(25.0%)		
With B_1+								
A	28	12	+1.6				37 cases	13 cases
B	13	9	+2.1				81% gained weight	85% gained weight
C	9	7	+1.7					50% improved
Total	50	9.1	+1.8	3.0 (72.0%)	3.5 (20.0%)	(8.0%)		

*Kindly supplied by Merck and Co., Rahway, N. J.

A study of our data brings out several interesting points. There are three respects in which the vitamin-treated group show improvement over the control series; namely, the degree of the reduction in pulse rate during the period of preparation, the number of patients who gained weight and whose appetite increased, and the length of time required for adequate preoperative preparation.

These findings are in agreement with the known effect of vitamin B_1 on the cardiovascular and gastrointestinal systems, if some degree of B deficiency exists. That vitamin B_1 or the B complex has no direct antithyrogenic action in man is shown by the fact that the two series showed no significant differences in the lowering of the basal metabolic rate before operation, nor in the severity of the postoperative reaction.

A point worth emphasizing is the fact that where improvement was noted in the vitamin-treated series it was most marked in the more toxic group. These are the patients in whom an avitaminosis is most apt to develop, and who in consequence should be most benefited by vitamin therapy. The milder cases of hyperthyroidism may not have developed any vitamin deficiency and it is well known that vitamin B_1 produces no effect on the normal gastrointestinal tract and normal cardiovascular system.

SUMMARY

It is our opinion that the severely thyrotoxic patients have been benefited by the administration of vitamin B_1. Not only is the nutritional state improved, as evidenced by increased appetite and weight gain, but also patients with severe hyperthyroidism can be adequately prepared for operation in a shorter time. That the vitamin treated cases showed a greater fall in heart rate during the preparatory period suggests that the tachycardia may have been due, in part, to a vitamin B deficiency and, therefore, was improved by the administration of adequate amounts of this vitamin.

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NONOPERATIVE TREATMENT OF PERFORATED GASTRIC ULCER WITH GENERALIZED PERITONITIS BY CONTINUOUS GASTRIC SIPHONAGE

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THE thesis of this comment is that surgery and surgeons not only "must" but "will," in the natural development of things, broaden their field of action to encompass all technical processes and new developments that come into immediate relation with surgical problems. In this perspective this paper affirms that the surgeon is today, in the course of his natural development, taking into his armamentarium a more comprehensive knowledge and use of such recent innovations as: (1) the Wangensteen nasal suction siphonage technique in the nonsurgical management of general peritonitis arising from ruptured appendix, perforated peptic ulcers, and bowel perforations and in postoperative peritonitis; (2) vitamin therapy for the deficiency diseases developing postoperatively in cases having a long convalescence and relative or absolute functional intestinal incompetency; (3) possible future hormonal, humoral, and chemical perspectives on and management of certain types of malignancies, hypertension, vascular spasms, and autonomic nervous system dysfunctions. In another way of saying it, surgery and surgeons, in their natural development, will acquire an insight into and a facility with related endocrine, chemical, physiologic, and physical data, as these features are established in more and more immediate relation to the so-called strictly surgical problem. The surgical ability and judgment necessary to the decision to operate connote equal ability and facility to not operate and to manage nonoperatively.

CASE REPORT

White male, aged 33 years, farmer, was admitted to University Hospital, Oklahoma City, Nov. 3, 1937, complaining of pain in stomach, nausea and vomiting, and bloating. History of attacks of dull, burning, epigastric pain, coming on three or four hours after eating, often referred to the chest area. Attacks lasted from a few days to two or three weeks, with pain-free periods of from one month to one year. The pain was usually relieved by taking food and also by taking sodium bicarbonate, although this usually caused vomiting and for that reason was rarely taken. Patient stated that he was often compelled to leave his work to obtain food which seemed to "expand" his stomach and relieve the pain. This pain seemed to be increased by exertion, excitement, or jarring. History of severe attack about two years ago which subsided after about two weeks. Present attack

Received for publication, June 13, 1938.

began about three weeks prior to admission to the hospital, characterized by almost continuous right subcostal and epigastric cramping, malaise, and some diarrhea. The pain during this attack was aggravated by taking food of any kind. There was some relief obtained by taking sodium bicarbonate, which was followed by vomiting. On the evening of Nov. 1, 1937, two days before admission, patient was stricken with a sudden sharp excruciating pain in the epigastrium and right subcostal area which was so severe that he was unable to lie down. Sodium bicarbonate was taken with no relief. Epsom salts also gave no relief. Morphine was finally administered by local physician with some relief.

On admission to hospital, examination revealed a dehydrated, emaciated, and apparently extremely ill patient. Temperature, 100.8°; pulse, 120; respiration, 24. Lips dry and cracked. Tongue very dry and covered with heavy dry mucus. Chest

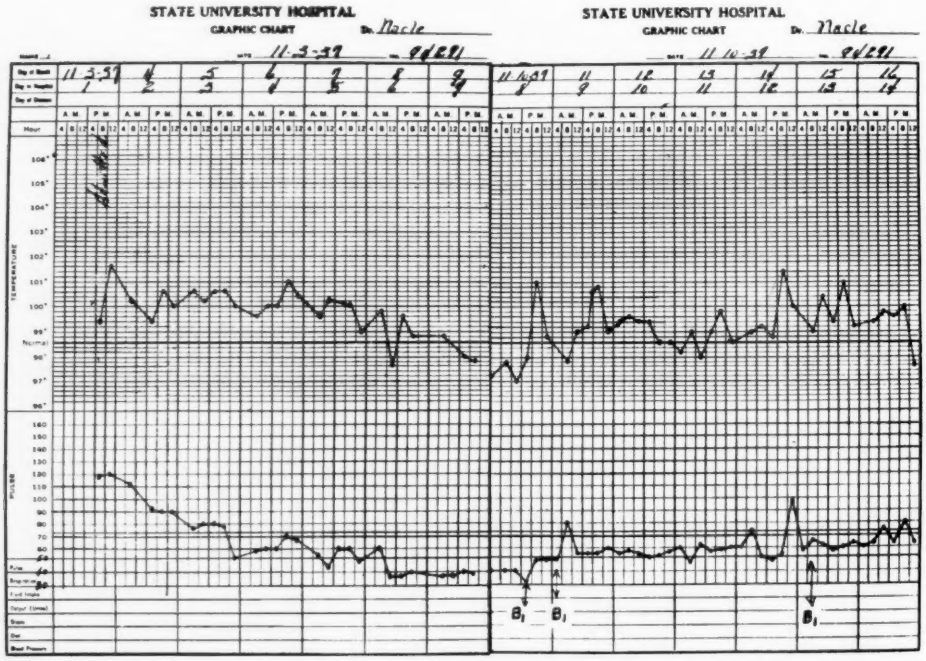

Fig. 1.—Shows a drop in pulse rate to a normal of 80 on the fifth day and a pronounced bradycardia of 36 to 40 beats per minute, beginning on the eighth day of illness.

and heart negative. There was uniform distention, shifting dullness, and flatness in each flank. Abdomen quiet, tympanitic in midportion and in suprahepatic area, with resonance above and flatness of liver below. Rebound and palpation tenderness generalized, more marked in right upper quadrant. *Laboratory:* Urine negative. Blood: Hemoglobin 15 gm. RBC 4,450,000; WBC 5,150, with 80 per cent neutrophiles. Widal, negative. Stools negative for typhoid. X-ray: Interpreted as "free air in abdominal cavity, some of which is separating liver from right diaphragm. Multiple fluid levels throughout. Small intestine loops are considerably dilated." Fluroscopic examination sixteen days later showed "stomach considerably distended by gas, deep large peristaltic waves, barium empties readily into duodenum; upper intestinal loops are moderately dilated. Large niche in the lesser curvature at the pylorus. Duodenal cap is normal. Stomach is empty in two hours. At six hours, terminal ileum is filled. X-ray findings are consistent with pyloric ulcer without

Fig. 2.—Reveals gas under both diaphragms, fluid levels in the stomach and many loops of the intestine, distention of the bowel with "fluid mirrors," "separation effect" of intraperitoneal fluid. This x-ray is definitely characteristic of perforated hollow viscus.

Fig. 3.—Shows x-ray after complete recovery, defining presence of large pyloric ulcer.

mechanical obstruction and residual paralytic ileus.'' *Diagnosis:* (1) Perforated peptic ulcer of forty-eight hours' duration with generalized peritonitis and intestinal ileus; (2) bradycardia, dicrotism, palpable at radial artery, which is not due to typhoid fever; (3) inanition with vitamin B_1 deficiency; (4) proved pyloric ulcer.

Management: Institution of management recommended by Wangensteen; namely, continuous nasal suction siphonage for gastric and intestinal decompression, parenteral fluids, interdiction of everything by mouth. Dramatic clinical improvement in forty-eight hours; abdomen generally soft, distention markedly reduced, hippocratic facies had disappeared, pulse and temperature reduced. Bradycardia of 36 manifested itself on the sixth day; was definite and sustained for two days thereafter, until the first administration of vitamin B_1. Patient was sustained for the first two weeks entirely by parenteral fluids, saline, glucose, Hartman's solutions. Synthetic vitamin B_1 in doses of 2,000 international units was given hypodermically. Surgical closure of perforation was not attempted and it was not necessary to drain residual abscesses. Subsequent convalescence uneventful.

COMMENT

It is to be noted in the history that for three weeks prior to admission to the hospital the patient had been taking soda for the relief of epigastric pain, had been vomiting, and had been unable to retain a normal diet. Nothing was given by mouth for the first fourteen days in the hospital. Nasal siphon was in continuous use for the first ten days in the hospital. It is believed by the writer that this further depleted the patient of all vitamin B_1 reserve and precipitated the bradycardia noted Nov. 8, 1937. This position is seemingly well taken, in the light of the remarkable and immediate remedial effect noted with each administration of synthetic vitamin B_1 hypodermically. Another effect of the vitamin B_1 is possibly the improved sedimentation rate. The writer evaluates this change in sedimentation time as an improvement in the vitality of the patient. In this relation a tenable hypothesis is that the sedimentation time is a function of the ''charge'' on the surface of the red blood cells, which, if reduced, permits a rapid falling out of suspension of the red blood cells and which, if ''normal'' or increased, holds the cells in separation and hence in suspension.

Early surgical closure of the perforation of the stomach and duodenum is always in order and surgical closure must be free from leakage. This is aided materially by maintaining continuous evacuation of the stomach and duodenum with suction siphonage. A point this paper wishes to emphasize is that it is not the hole in the stomach per se, but the leakage through the hole in the stomach that causes and maintains the fatal peritonitis in these cases and that even in cases that seem too late for surgical closure of the perforation the prognosis is materially improved by maintaining a continuously empty stomach and duodenum. This may be effected only by the continuous nasal suction siphonage.

The popularization of the nasal suction siphon decompression of the upper reaches of the intestinal tract will probably stand out in future

years as the event signalling a reorientation in surgical thinking to a more physiologic and more dynamic pattern than previously. Proficient technicians will always be appreciated, but surely in the coming years, if surgery is to be advanced, more stress and emphasis must be placed upon its physiologic aspects. The surgery of tomorrow will be defined by minds that are challenged by a problem and that do not abate their efforts until a practical solution is uncovered. In the same perspective it appears certain that medical knowledge is advanced more by the elaborate and painstaking study of individual case problems than by statistical reviews.

CONCLUSION

The instance of a patient with acute perforation of forty hours' standing of a gastric ulcer into the free peritoneal cavity successfully treated without operation by continuous suction applied to an indwelling duodenal tube is reported. The early closure of all perforations of the gastrointestinal canal by open operation is insisted upon. The great value of decompression by gastric and intestinal siphonage in late cases is discussed.

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ACIDITY OF GASTRIC CONTENTS AFTER EXCISION OF THE ANTRAL MUCOSA*

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INCONTROVERTIBLE proof that the antral mucosa is of physiologic importance in the maintenance of the intragastric or chemical phase of gastric secretion has not as yet been presented. The suggestion that when food comes into contact with the antral mucosa the fundic mucosa is stimulated to secrete acid is an interesting but unproved hypothesis.

Interest in the physiology of the intragastric phase of gastric secretion was stimulated by the work of Edkins in 1906. He found that an extract of the antral mucosa contained an active substance, which, when injected intravenously into dogs, stimulated secretion of gastric juice. The results of subsequent investigations would indicate that histamine was the active principle in the extracts employed by Edkins.

Since Edkins' work there has been a more or less fruitless search for some hormonal substance which is contained in or liberated by the antral mucosa and which stimulates gastric secretion. Many active extracts have been prepared, yet the subject of hormonal stimulation of gastric secretion is still confused. Nevertheless, a true gastric secretin may exist.

The somewhat less complicated problem of whether or not the antral mucosa affects secretion in the fundus sufficiently to be physiologically important has been extensively studied. In one experimental approach food is placed in an isolated antral pouch, and fundic secretion is analyzed for acid. Edkins and Tweedy, Lim, Ivy, and McCarthy, Johnson, Babkin, and Sawitsch and Zeliony found acid secretion in the fundus. Priestley and Mann did not find satisfactory evidence that food in an antral pouch stimulated acid secretion in the fundus.

In another approach to the problem, whole stomach pouches were prepared, some with and some without the antrum of the stomach. Analysis of the gastric contents was then done in the usual fashion. Kim, Babkin, and Wilhelmj, O'Brien, and Hill obtained decreased acid secretion in those whole stomach pouches from which the antral portion had been removed.

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*Abstract of thesis submitted to the Faculty of the Graduate School of the University of Minnesota in partial fulfillment of the requirement for the degree of M. S. in Surgery.

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Received for publication, June 14, 1938.

A third method of study involves resection of the antrum and performance of gastroenterostomy in the same way that partial gastrectomy is performed on the human being. With test meals, Smidt, Babkin, Wilhelmj, O'Brien and Hill, and Klein found reduction in acid secretion after resection of the antrum. Portis and Portis, Steinberg, Brougher and Vidgoff, Priestley and Mann, Fauley, Strauss, and Ivy, and Shapiro and Berg obtained either inconclusive evidence of reduction in acidity, or only temporary hypoacidity.

Gastroenterostomy permits more regurgitation of intestinal contents than is found in the normal stomach. In all of the experimental methods mentioned, the muscular and probably the nervous continuity of the

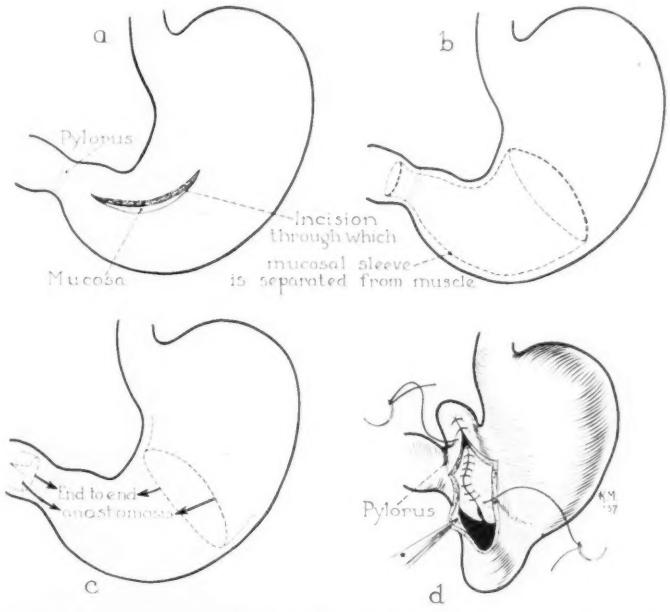

Fig. 1.—Diagrammatic representation of steps in the operation for the removal of the antral mucosa; *a*, longitudinal incision extending down to mucosa; *b*, sleeve of mucosa that is excised; *c*, end-to-end anastomosis; *d*, completion of anastomosis.

stomach and the intestine is interrupted. As both of these factors make experimental results difficult to interpret, an attempt was made in this study to excise the antral mucosa without causing excessive regurgitation of intestinal fluid and with as little interruption of gastrointestinal continuity as possible.

METHOD

Operation.—A longitudinal incision 6 to 8 cm. long was made on the anterior surface of the distal portion of the stomach. This incision terminated 2 cm. proximal to the pyloric muscle and extended down to the mucosa. By blunt dissection the mucosa was separated from the muscularis distally into the first centimeter of the duodenum and

proximally till the increase in fibrous attachment to the muscularis indicated that fundic mucosa had been reached. The cuff of antral mucosa thus freed from the muscularis was excised and the edge of fundic mucosa was anastomosed to the edge of the duodenal mucosa. The longitudinal incision in the muscularis was closed transversely in order to prevent tension on the mucosa (Fig. 1). By this procedure the pyloric mucosa is removed; the pyloric muscle remains intact and in control of the outlet of the stomach, and only mucosal continuity is interrupted.

To determine the effect of the operation on the emptying time of the stomach, roentgenoscopic studies with a standard (throughout the study) barium meal were made before and after operation.

As the trauma of operation is said to diminish acid secretion for a time, analyses of gastric contents were done, with subcutaneous injection of histamine as the stimulus, before and after operation.

A standard (throughout the study) ground meat and water test meal was used to determine the secretory response to food in the presence of and in the absence of the antral mucosa. Analyses of the gastric contents with histamine and with the meat meal were made one to four months after operation, and analyses with the meat meal were repeated eight to eleven months after operation.

Ten to twelve months after operation the dogs were killed; the stomachs were examined grossly and specimens of tissue were cut from two places in the mucosal suture line in each stomach for histologic study of the mucosa on each side of the anastomosis.

RESULTS

Within one month after the operation, the animals were well nourished and in good condition. The emptying time of the stomach of different animals varied between four and seven hours before operation. One to three months after operation the emptying time varied between four and eight hours. Therefore, the gastric emptying time after operation was considered to be within the limits of normal variation.

Analyses of the gastric contents after stimulation with histamine revealed little significant change in acidity curves after resection of the antral mucosa (Fig. 2 and Table I). Hence the capacity of the fundic mucosa to secrete acid after strong stimulation apparently was not affected by the operation.

Curves of acidity of the gastric contents obtained by analysis after administration of the meat meal were much lower than the normal acidity curves in four dogs one to four months after operation (Fig. 3 and Tables II and III). In two dogs these acidity curves were only

a little lower than the normal acidity curves determined before operation (Fig. 4 and Tables II and III). Eight to eleven months after operation the dogs that had a much lower acidity after operation than they did before showed some slight additional lowering of acidity. One of the dogs that had but little lowering of acidity of the gastric contents im-

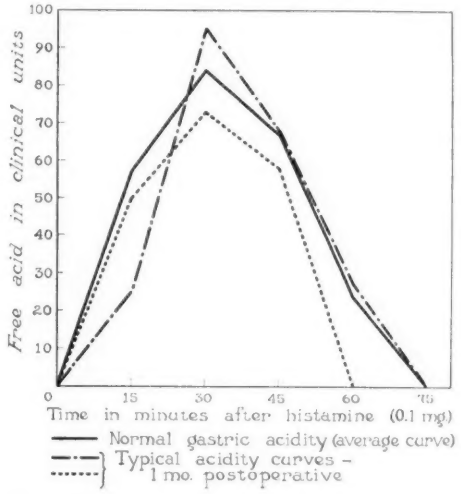

Fig. 2.—Results of analyses of gastric contents after injection of histamine (Dog 1).

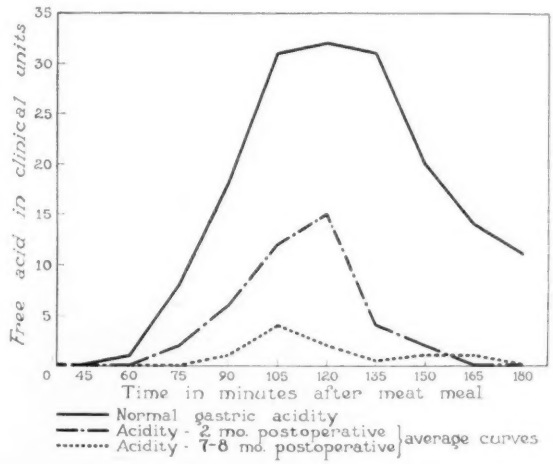

Fig. 3.—Results of analyses of gastric contents after administration of meat test meal and total excision of antral mucosa (Dog 1).

mediately after operation also showed a little additional lowering of the acidity in eight to eleven months after operation.

On gross examination of the stomachs immediately after the dogs were killed, the mucosa appeared to be normal and the pyloric outlet appeared to be of normal size and contractibility. Sections prepared from the tissue at the mucosal anastomosis were examined microscopically. Fundic mucosa was found adjacent to duodenal mucosa in the stomachs

TABLE I

ANALYSES OF GASTRIC CONTENTS AFTER ADMINISTRATION OF HISTAMINE (DOG 1)

MINUTES AFTER ADMINISTRATION OF HISTAMINE	0	15	30	45	60	75
Average titration figures (clinical units) in normal dog	0	57	84	67	24	0
Titration figures in same dog 1 month after resection of antral mucosa	0	25	95	68	27	0
Two typical curves	0	50	73	58	0	0

of the four dogs with marked lowering of gastric acidity after operation (Fig. 5). A very small amount of antral mucosa was found on the gastric side of the mucosal anastomosis in the two dogs with only slight reduction of acidity after operation.

TABLE II

SUMMARY OF ANALYSES OF GASTRIC CONTENTS AFTER MEAT MEALS

DOG	DESCRIPTION	NORMAL			1 TO 3 MO. AFTER RESECTION			7 TO 11 MO. AFTER RESECTION		
		NUMBER OF ANALYSES	RANGE OF MAXIMAL VALUE FOR FREE ACIDITY (CLINICAL UNITS)	AVERAGE MAXIMAL VALUE FOR FREE ACIDITY (CLINICAL UNITS)	NUMBER OF ANALYSES	RANGE OF MAXIMAL VALUE FOR FREE ACIDITY (CLINICAL UNITS)	AVERAGE MAXIMAL VALUE FOR FREE ACIDITY (CLINICAL UNITS)	NUMBER OF ANALYSES	RANGE OF MAXIMAL VALUE FOR FREE ACIDITY (CLINICAL UNITS)	AVERAGE MAXIMAL VALUE FOR FREE ACIDITY (CLINICAL UNITS)
1	Total resection of antral mucosa	15	60 to 15	32	16	37 to 10	15	14	20 to 0	4
2	Total resection of antral mucosa	12	90 to 42	46	15	57 to 0	18	13	35 to 0	11
3	Total resection of antral mucosa	12	65 to 27	30	17	45 to 0	16	14	25 to 0	7
4	Total resection of antral mucosa	18	60 to 25	31	17	45 to 0	9			
5	Subtotal resection of antral mucosa	16	63 to 30	40	16	90 to 45	46			
6	Subtotal resection of antral mucosa	16	63 to 32	38	14	48 to 20	29	14	45 to 10	15

COMMENT

Although direct proof is lacking, it seems logical to infer that in the absence of abnormal coloration of gastric contents by bile, with a normal gastric emptying time, with the pyloric outlet of normal size, and with the pyloric muscle intact, regurgitation of intestinal contents would not be increased very much if at all after operation. A normal acidity of the gastric contents after stimulation with histamine also is some indication that intestinal contents did not neutralize and dilute gastric secretion to an abnormal degree after operation.

If the trauma of operation diminished the capacity of the fundic mucosa to secrete hydrochloric acid, one would expect lowering of the acidity of the gastric contents following stimulation with histamine as well as after a meat test meal. However, the acidity of the gastric con-

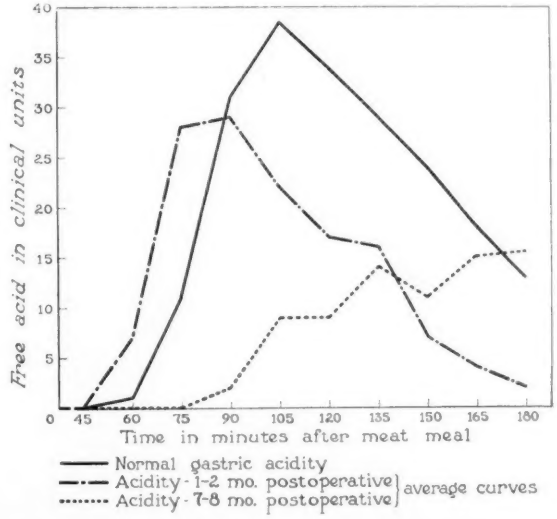

Fig. 4.—Results of analyses of gastric contents after administration of meat test meal and subtotal excision of antral mucosa (Dog 6).

Fig. 5.—Section from mucosal anastomosis; animal whose acidity curves are shown in Fig. 3; fundal mucosa may be seen on the right and duodenal mucosa on the left (Dog 1).

tents following stimulation with histamine was not lowered after operation. Therefore, dilution and neutralization of gastric secretion by excessive quantities of intestinal secretion, and the trauma of the operative procedure, cannot well account for the hypoacidity, with a meat test meal, that was present after removal of the antral mucosa. Yet in some manner the normal stimulus to acid secretion that is usually associated

TABLE III

ANALYSIS OF GASTRIC CONTENTS AFTER ADMINISTRATION OF MEAT MEAL; AVERAGE TITRATION FIGURES EXPRESSED AS CLINICAL UNITS

TIME IN MINUTES AFTER ADMINISTRATION OF MEAT MEAL		0	45	60	75	90	105	120	135	150	165	180
Dog 1 (see Fig. 3 and Table II) total resection of antral mucosa	Normal dog	0	0	1	8	18	31	32	31	20	14	11
	1 to 2 mo. after resection of antral mucosa	0	0	0	2	6	12	15	4	2	0	0
	7 to 8 mo. after resection of antral mucosa	0	0	0	0	1	4	2	0.5	1	1	0
Dog 6 (see Fig. 4 and Table II) subtotal resection of antral mucosa	Normal dog	0	0	1	11	31	38	34	29	24	18	13
	1 to 2 mo. after resection of antral mucosa	0	0	7	28	29	22	17	16	7	4	2
	7 to 8 mo. after resection of antral mucosa	0	0	0	0	2	9	9	14	11	15	15

with the presence of food in the stomach was appreciably diminished after the removal of the antral mucosa. The same operation was performed on two dogs, except that a very small portion of the antral mucosa was left accidentally in the stomach. Marked hypoacidity did not occur in these two animals.

A cursory examination of the results of these experiments would appear to indicate that the loss of the antral mucosa, with the stomach otherwise intact, impairs the normal intragastric stimulation of acid secretion and that these results are due to an absence of a hormone produced in the pyloric mucosa. However, two considerations of the data seriously invalidate the conclusions that the existence of a pyloric hormone has been proved by these experiments. It does not appear probable that the small amount of pyloric mucosal remnant which was certainly less than 1 per cent of the total amount of antral mucosa, left in two of the animals, was the cause of a hypoacidity's not developing on the basis that this small amount of mucosa produced an adequate amount of hormone. A serious objection for accepting these results as proof that a pyloric hormone exists is the fact that the hypoacidity developed progressively and was greater eleven months after operation than it was at the end of the first month. It should be noted that, according to the results of the histamine test, the secretory tone of the fundic mucosa was maintained throughout the period of observation.

As the number of animals used in this study is small, definite conclusions are not justified. Yet the close similarity of the results in different animals suggests that these observations may be of some physiologic significance. More extensive study of a larger group of animals would be necessary to avoid some of the possible sources of

error in these studies and to give more statistical significance to the experimental results. Different types of test meals also should be employed as the ideal test meal has not as yet been devised for this type of investigation.

SUMMARY

Total excision of the antral mucosa was accomplished without interrupting the muscular and, in all probability, the nervous continuity of the gastrointestinal tract, and without obvious increase in regurgitation of intestinal contents into the stomach. Complete excision of the antral mucosa did not change the secretory response to the histamine test, but was followed by marked hypoacidity, as determined by a meat test meal. Further investigation is necessary before the results obtained can be ascribed to the absence of a pyloric hormone. However, the method used does offer possibilities of proving whether or not such a hormone exists.

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WANDERING SPLEEN WITH TORSION OF THE PEDICLE

A Patient Treated by Splenectomy With Alarming Events During Convalescence

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WANDERING spleen with torsion of the pedicle is of relatively infrequent occurrence. Abell[1] collected 95 case reports and added 2 cases of his own to this series. Of this number only 1 patient was under 10 years of age, a male child 6 years old, operated upon by Southam,[2] of England, in 1921. This boy was seized with a sudden, sharp pain in the right iliac fossa and was thought to have an acutely inflamed appendix. Laparotomy revealed a large, congested spleen in the right iliac fossa twisted two and one-half times on an elongated pedicle. Splenectomy was done and the boy was discharged recovered nineteen days after operation.

In August, 1934, Percy[3] reported a similar case in a boy 10 years old. Motley[4] added a third case in a boy 8 years of age. These children also recovered after splenectomy. These are the only case reports we have been able to find in which the patients were under 10 years of age.

Because of the rare opportunity to study this condition in a child, the following case from our records is reported.

Case Report.—No. 40774. F. D., a white female child, aged 8 years, was admitted to the hospital Nov. 23, 1937 at four o'clock in the afternoon. Her mother stated that she had always been well. In early childhood she had measles and chicken pox but no serious illnesses or operations. Her bowels had always been sluggish. For a week before admission she had an upper respiratory infection with mildly productive cough. Two days before entry she complained of pain in the region of the umbilicus associated with nausea and vomiting. Twenty-four hours later the pain became localized in the right lower quadrant. Anorexia, nausea, and vomiting persisted. The severity of the pain increased and spread throughout the entire abdomen. She was seen by Dr. John McNamara, of Taunton. Upon his first examination he found the mass centrally located in the lower abdomen. From its smooth surface, its firm texture and mobility, he suspected a cyst of pelvic origin. Then he felt an indentation on one border which at that examination suggested the spleen. At a later hour, however, the mass had shifted to the right iliac fossa. Finally, believing it to be an appendiceal abscess involving the omentum, he sent the patient to the hospital.

On admission the child's temperature was 101.4°, her pulse 120, and respirations 24. She looked moderately ill, and coughed frequently as a result of the upper respiratory infection. The lungs were clear and resonant. The heart was normal. No murmurs were heard. There was a mass in the right lower abdomen, smooth,

Received for publication, June 16, 1938.

dense, movable, and tender. The impression of the examiner was that of either an omental abscess of appendiceal origin or an ovarian cyst with twisted pedicle. At this time the white count was 24,000.

Laparotomy was performed eight hours after admission. Upon opening the peritoneum, serosanguineous fluid escaped. A large, bluish-black mass occupied the right lower quadrant of the abdomen. This was identified as the spleen several times its normal size. It was not adherent and was readily delivered through an enlarged incision. The pedicle was about 2½ cm. in diameter, and was twisted three times clockwise on its axis. The veins were thrombosed. The pedicle was clamped, ligated,

Fig. 1.—The spleen.

and the spleen was removed. The appendix was normal and was left in situ. There was no evidence of thrombosis of the mesentery.

The appearance of the spleen is seen in Fig. 1. It weighed 425 gm. as compared with 110 gm., the average normal weight of the spleen in a child 8 years of age. Except for its enlargement, it was normal in configuration. The capsule was smooth and darker than normal in color. The pedicle was cordlike instead of the usual membranous type. Cut section revealed a firm pulp, dark maroon in color, with numerous hemorrhagic areas. Microscopic examination showed tremendous engorgement of the pulp with areas of infarction. The Malpighian corpuscles were not readily discernible. The diagnosis was strangulation of the spleen due to torsion of the pedicle.

After operation the patient coughed frequently, raising small amounts of white mucus. She vomited at intervals almost all fluids taken. Acetone was present in a specimen of urine. The CO_2 combining power was not determined because of a defective apparatus. Therefore, to combat acidosis, parenteral fluids were administered. No inflammatory changes in the wound were noted.

X-ray examination of the chest Nov. 29 showed no evidence of lung pathology.

For a week after operation her condition remained about the same. At 4:00 A.M. on Nov. 30, after an attack of coughing, several feet of small intestine eviscerated through the incision. Under chloroform anesthesia the intestine was replaced and the wound closed with through-and-through sutures of silk-worm gut. A transfusion of 300 c.c. of citrated blood was given and gastric drainage employed. The patient improved rapidly after this intervention. She retained all fluids and bowel function was restored.

Blood studies carried out during convalescence showed the following:

	ERYTHROCYTES	LEUCOCYTES	HEMOGLOBIN	NEUTROPHILES	LYMPHOCYTES	LARGE MONONUCLEARS	EOSINOPHILES
Nov. 23		24,000					
Nov. 24	4,490,000	24,800	80%	82%	8%	10%	
Nov. 25		22,400					
Nov. 26	3,990,000	11,900	72%	61%	32%	5%	2%
Nov. 27		15,100					
Nov. 29		28,700					
Nov. 30	3,540,000		65%	81%	16%	3%	
Dec. 1		24,600					
Dec. 3		11,900					
Dec. 8	3,690,000	7,800					
Dec. 9		7,700					
Dec. 10	3,780,000	8,900					
Dec. 11		8,800					
Dec. 13	3,870,000	8,200					
Dec. 14		8,900					
Dec. 15	3,850,000	9,300					
Dec. 16		9,100					
Dec. 17	4,250,000	7,500					
Dec. 18		6,200					

The number of blood platelets rose from 180,000 to 270,000 from Dec. 3 to Dec. 18. Two analyses of the smear showed anisocytosis and some polychromasia. The blood Kahn test was negative. The bleeding time was 1 to 1½ minutes.

The blood sugar was 105 mg.; the nonprotein nitrogen following the first operation was 30 mg. On the day before evisceration occurred, it rose to 49.8 mg.

Analysis of seven specimens of urine was negative for acetone.

This patient was discharged recovered Dec. 19, 1937, twenty-six days after admission. She felt very well for five days. Then on Dec. 24 she began to complain of sharp, colicky pain in the left upper quadrant of the abdomen. During the day she vomited frequently and had complete obstipation. Since her condition did not improve, she was readmitted to the hospital.

There it was found that the abdomen was distended and tympanitic. There was tenderness to the left of the umbilicus. Peristalsis was visible and borborygmus marked. Examination of the urine was negative. The blood picture was the same as that of the preceding week: 77 per cent hemoglobin; 3,810,000 erythrocytes; 7,900 leucocytes; 61 per cent neutrophiles; 32 per cent lymphocytes; 5 per cent large mononuclears; 1 per cent eosinophiles; and 1 per cent basophiles. The impression of the examiner was acute intestinal obstruction.

The abdomen was reopened. A loop of small bowel about 35 cm. in length, purplish in color, was found obstructed by a dense fibrous band. This was severed and immediately the color of the affected bowel improved. The abdomen was then closed.

This convalescence was uneventful. Examination of the blood showed the following:

	ERYTHRO-CYTES	LEUCO-CYTES	HEMO-GLOBIN	NEUTRO-PHILES	LYMPHO-CYTES	LARGE MONO-NUCLEARS	EOSINO-PHILES	BASO-PHILES
Dec. 28	3,810,000	7,900	77%	61%	32%	5%	1%	1%
Dec. 31	3,980,000	8,000	80%	56%	34%	8%	2%	
Jan. 3	3,760,000	6,700	77%	54%	39%	3%	3%	1%
Jan. 6	4,100,000	9,700	78%	42%	52%	5%	1%	
Jan. 10	3,990,000	11,800	79%	35%	61%	3%	1%	
Jan. 12		8,800						
Jan. 13	3,990,000	8,700	80%	28%	68%	4%		

The blood platelet count ranged from 230,000 to 270,000. In two specimens there was slight anisocytosis. The only other abnormality noted was a steady decrease in the percentage of the neutrophiles with a corresponding rise in the percentage of the lymphocytes.

This patient was discharged Jan. 15, 1938, three weeks after operation, and has remained well.

DISCUSSION

Wandering spleen usually remains symptomless until the pedicle undergoes torsion. Colic of mild type, perhaps due to partial twists of the pedicle, and digestive and circulatory disturbances due to pressure on other organs have been reported. Pain, nausea, vomiting, elevation of pulse, temperature, and high leucocyte count usually accompany an acute attack of strangulation. Besides complete torsion of the pedicle, the bowel may become adherent to or compressed by the twisted pedicle.

Splenomegaly is not considered a factor in elongation of the pedicle, for wandering spleen with twisted pedicle has not been reported in cases of Banti's or Gaucher's disease, leucemia, or hemolytic jaundice. It occurs most frequently in multiparae. When torsion of the spleen occurs during pregnancy or the puerperium, the prognosis is unfavorable, the mortality rate being 41.7 per cent. In Abell's series of 97 cases, a history of malaria was given in 28. Torsion ranged from one-half to six complete turns. The longest pedicle described measured 25 cm.

A study of the embryology of the spleen may throw some light upon these cases of wide excursion of this organ. Normally the location of the spleen is fairly constant. It is situated in the upper left posterior portion of the abdomen in juxtaposition with the diaphragm. The average adult spleen weighs 160 gm. and measures 12 by 7 by 4 cm.

The posterior border is smooth; the anterior edge is thin and has a number of grooves which from a diagnostic point of view are very important in hypertrophy of the organ. The spleen with its pedicle,

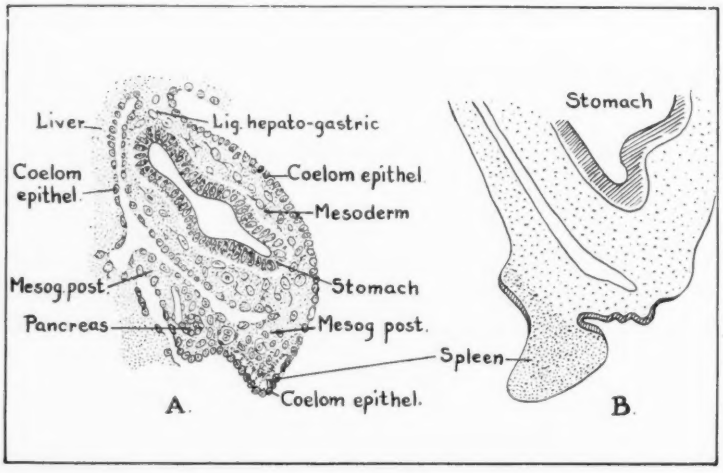

Fig. 2.—Two stages in the early development of the spleen; *A*, from an embryo of 10.5 mm. (Kollmann); *B*, from a 20 mm. embryo (Toukoff). (Redrawn from Prentiss: Text Book of Embryology, W. B. Saunders Company, Philadelphia, 1915.)

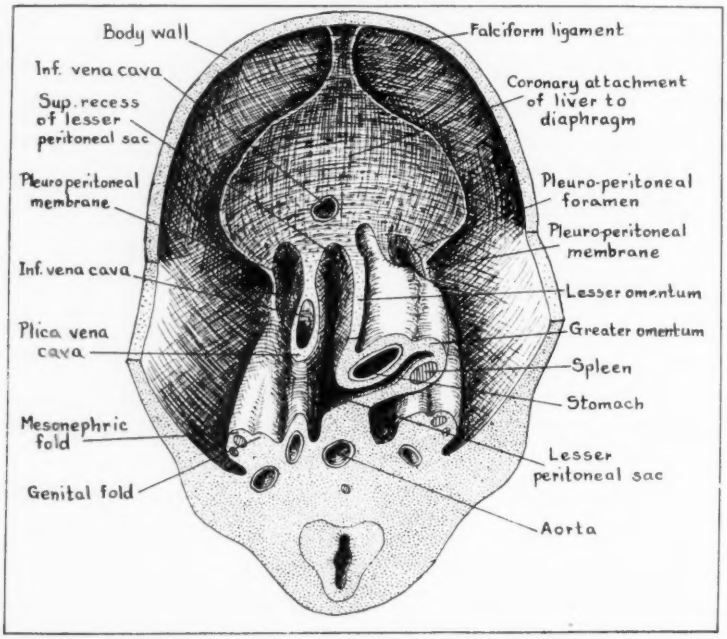

Fig. 3.—A diagrammatic ventral view of the middle third of an embryo 12 to 15 mm. long. (Redrawn from Prentiss: Text Book of Embryology, W. B. Saunders Company, Philadelphia, 1915.)

usually short, may be said to resemble a mushroom on its stalk. Normally the head is free and entirely covered with peritoneum. The stalk or pedicle, however, is supported in position by the lienorenal and

gastrosplenic ligaments and below by the phrenicocolic ligament. The nerves, arteries, veins, and lymphatics which supply the spleen are all enclosed in this pedicle.

The spleen is mesodermal in origin. It appears first in a five-week human embryo (10 mm.) as a thickening of the dorsal mesogastrium when the cells of the peritoneal epithelium divide. It is located in the cranial portion of the greater omentum. See Fig. 2*A*.[5] By the time the embryo is 7½ weeks old (20 mm.), the spleen anlage has become fairly well demarcated. See Fig. 2*B*. The position of the spleen rela-

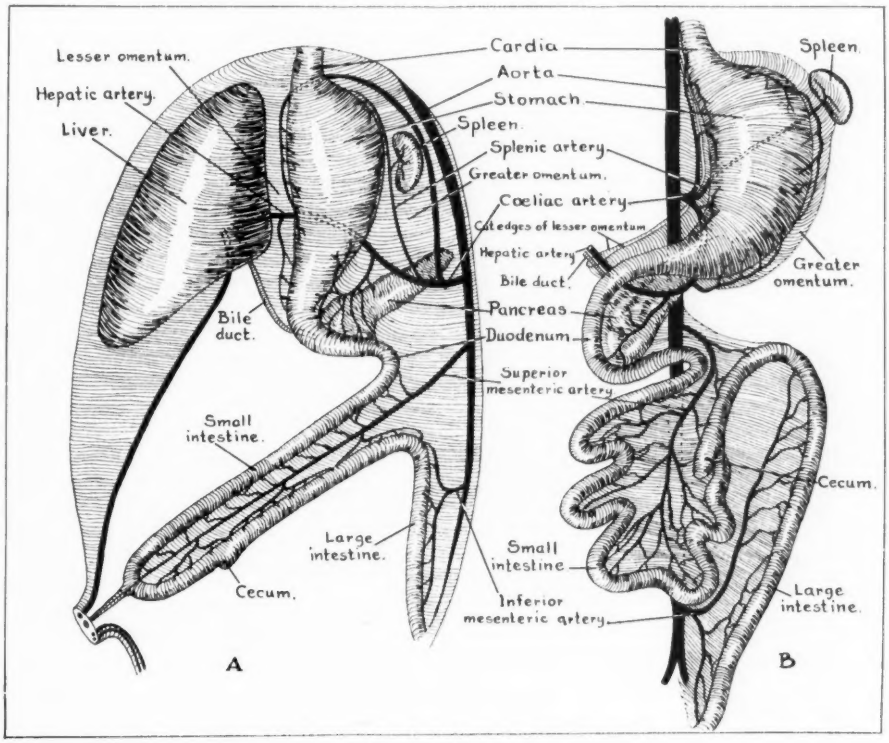

Fig. 4.—Schematic drawing showing early embryonic development of spleen in relation to other structures. (Redrawn from Sabotta and McMurrich: Atlas of Human Anatomy, Vol. II, G. E. Stechert and Company, New York, 1928.)

tive to omentum and stomach is clearly seen in Fig. 3,[6] which shows a section through stomach and spleen in an embryo 6 weeks old (12 to 15 mm.).

The septum transversum begins its caudal migration when the embryo is 4 weeks old (7 mm.). With its descent there is constant shifting of all structures. The body cavities increase in size and the liver grows enormously. The descent of the stomach begins when the embryo is 5 weeks old (10 mm.), just when the spleen anlage is making its appearance. It descends rapidly and in two weeks has reached its permanent position below the diaphragm. When the dorsal wall of the stomach descends and rotates to the left, the greater omentum is carried

with it. Since the spleen develops in the greater omentum, its position is affected by the rotating stomach in particular. If attachments between the stomach and the spleen are thin or meager, the spleen gradually increases its range of motion.

When the embryo is six weeks old, the intestinal loop enters the umbilical cord and remains there for three or four weeks until the embryo is about 42 mm. in length (ninth or tenth week). Fig. 4*A*[7] is a lateral view of the stomach and spleen in a 6-week embryo (12 to 15 mm.) just before the gut enters the umbilical cord. Fig. 4*B* is an anterior view of these organs after herniation of the gut. The pyloric end of the stomach has rotated more to the right and the spleen has assumed a higher position relative to the greater curvature of the stomach. The intestines have elongated with torsion on the mesentery and shifting of all structures.

DIAGNOSIS AND TREATMENT

Since the spleen may migrate to any part of the peritoneal cavity, the correct diagnosis is rarely made on clinical examination. The condition is often interpreted as appendicitis with peritonitis, abdominal tumor, or ovarian cyst with twisted pedicle. Laparotomy is done for an acute abdominal crisis and the engorged spleen with twisted pedicle is revealed.

Conservative measures are not indicated. Operations which attempt to conserve the spleen which has already sustained damage to its blood vessels and has a tendency to wander from its normal position will afford only temporary relief, if any. These cases later show acute torsion and eventually have to be treated by splenectomy. The loss to the body of the function of the spleen has been offered as an argument in favor of conservative treatment. But other tissues rich in reticuloendothelial cells compensate for the loss of the spleen's function of hematopoiesis.[1]

In our case the blood study was carried out through convalescence. The final hospital blood examination Jan. 13, 1938, showed 80 per cent hemoglobin, 3,990,000 erythrocytes, and 8,700 leucocytes. The differential count, however, during the period of Dec. 28 to Jan. 13 showed a steady decrease in the number of neutrophiles and a corresponding rise in the number of lymphocytes.

This patient returned for a blood examination April 29, 1938. Her hemoglobin was 80 per cent, with 4,270,000 erythrocytes, and 9,000 leucocytes. The differential count still showed inversion of the number of neutrophiles and lymphocytes, with 35 per cent of the former and 62 per cent of the latter; 2 per cent eosinophiles and 1 per cent large mononuclears. The platelets numbered 200,000.

The bleeding time was 1 to 1½ minutes; clotting time (venous) 5 to 5½ minutes. The fragility test showed hemolysis beginning at 0.42

and ending at 0.28, as compared with the control fragility test, which began at 0.42 and ended at 0.32. Thus, for the most part, findings were normal. The child had gained 22 pounds and except for a tendency toward constipation had no complaints.

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ENDOMETRIAL TUMORS IN POSTCESAREAN ABDOMINAL LAPAROTOMY SCARS

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SOMEWHERE between the classifications of classical tumor pathology, physiologic hyperplasias, and embryonal maldevelopments, there exists a lesion known, for want of a better name, as endometriosis. This lesion consists of an infiltrative epithelial element resembling uterine mucosa in appearance, embedded in a connective tissue stroma, reinforced occasionally by smooth muscle tissue. The varying conceptions of its nature are reflected in the many names by which it has been called, including adenomyoma, indicating its supposed neoplastic character; endometriosis, indicating its diffuse hyperplastic character; peritonitis adenoides, indicating its supposed inflammatory character; mullerianoma, indicating its embryonic origin; and a number of other terms, each of which tends to emphasize one particular aspect of its numerous characteristics. Until its fundamental nature is definitely established, the simple descriptive term, endometrial tumor, is to be preferred as the least committal and yet most inclusive one by which to designate it, the term tumor in this connection being used in its widest sense.

Imperfect as is our knowledge of this lesion, there has grown up a voluminous literature on its clinical manifestations, pathology, and pathogenesis. The latter, particularly, is today still a matter of considerable controversy. To discuss all these subjects comprehensively is beyond the scope of this communication, which is intended to draw attention to an infrequent type of endometrial tumor occurring in postcesarean laparotomy scars. These tumors, because of the circumstances peculiar to their origin and location, may shed light on the general problem of the pathogenesis of similar tumors occurring in other parts of the body.

SITES OF ENDOMETRIAL TUMORS

A strong predilection exists for the localization of these tumors within or adjacent to the female genital organs, including the uterus, Fallopian tubes, ovaries, broad ligament, and the pelvic and lower abdominal peritoneum. Involvement of the serosal surfaces of the small intestine, appendix, colon, rectum, and bladder is found less commonly. Rela-

Received for publication, June 6, 1938.

tively rare are the extraperitoneal forms of endometriosis, of which there are recognized: a primary form occurring in the navel and groin; and a secondary form occurring in the abdominal wall and pelvic extraperitoneal tissues which have been exposed during operative procedures. It is the latter group of cases with which we are especially concerned in this review, particularly those instances in which the pregnant uterus had previously been opened during a cesarean section. The relative frequency of the various sites of endometrial implants, as reported by Keene and Kimbrough[1] in 119 cases of endometriosis, is as follows:

Ovary		110
Unilateral	63	
Bilateral	47	
Rectovaginal septum		6
Umbilicus and ovary		2
Laparotomy scar		1

INCIDENCE OF POSTCESAREAN ENDOMETRIAL IMPLANTS OF THE ABDOMINAL WALL

Since 1903, when Meyer[2] reported the first case of an endometrial implant in a laparotomy scar following a ventrosuspension of a nonpregnant uterus, numerous instances of this sequel to gynecological operations have been reported. Three years later, von Franqué[3] reported the first case in which the abdominal wall implantation followed the opening of the pregnant uterus. By 1925 Heaney[4] was able to collect 29 cases in which endometrial tumors formed in laparotomy scars. These were distributed as follows: 14 following ventrofixation, 2 following hysterectomy, 3 following pelvic operations, 2 following appendectomy, 1 following oophorectomy, and 7 following opening of pregnant uterus.

Hosoi and Meeker[5] in 1929 collected 87 cases of endometrial tumors in laparotomy scars of which 19 followed cesarean section. To date we have been able to collect a total of 53 cases* following the opening of the pregnant uterus. Most of these cases have been published in the form of individual case reports and short communications, with the result that the occurrence of such a sequel to cesarean operations has not been made generally familiar to surgeons of otherwise wide experience.[6] Doubtless, as general surgeons and gynecologists become more familiar with this condition, it will be seen to be a lesion of even greater frequency.

Our case is the following:

*We believe that one case attributed to Stavely by Danforth[38] is identical with the one attributed to Stavely by Burnham and Cullen.[46]

CASE REPORT

O. D. (Bronx Hospital No. 80596), a 19-year-old married woman, was admitted with a history that she had been delivered of a child at term by classical cesarean section eighteen months previously. Her postoperative course had been stormy with wound infection and healing by secondary intention. Several weeks thereafter she noticed a small cherry-sized nodule in the abdominal scar, slightly tender on pressure and exquisitely painful at each menstrual period. The nodule had gradually increased in size.

Fig. 1.

Physical examination revealed no abnormalities other than a slightly tender, walnut-sized, irreducible, hard nodule in the midline abdominal scar about 1 cm. below the level of the umbilicus. The nodule was fixed to the deep fascia but was not intimately adherent to the overlying skin.

In view of the characteristic history a diagnosis of endometrial tumor of a postcesarean laparotomy scar was made and excision performed. The tumor was found to be firmly attached to the fascia and to the subjacent parietal peritoneum as well, although there were no intraperitoneal structures adherent to the nodule. Palpation of the internal genitals through the peritoneal wound revealed no abnormalities. The tumor possessed no well-defined capsule so that a wide excision was performed in order to minimize the possibility of a recurrence.

On section the tumor was seen to be made up of radiating strands of hard fibrous tissue which extended into the surrounding areolar tissue. Between these strands there were islands of mucoid material. Microscopic examination by Dr. Joseph Felsen revealed the tumor to be "endometriosis in a fibrous stroma with areas of myxomatous degeneration and histiocytosis." (Fig. 1.)

Recovery was uneventful, the wound healing by primary union and with complete relief of symptoms.

CLINICAL FEATURES

Endometrial tumors of postcesarean laparotomy scars present a more or less characteristic clinical picture which should offer little diagnostic difficulty to the surgeon familiar with this lesion. Following a period varying from several weeks to several months after the original operation, the patient, a woman generally less than 35 years of age, becomes aware of a hard nodule in the scar. In a few isolated cases a much longer interval elapsed between the time of the original laparotomy and the appearance of symptoms attributable to the endometrial tumor. Pankow[7] and Mahle and MacCarty[8] report instances in which an interval of about twenty-five years elapsed between the time of the original laparotomies (appendicectomies) and the discovery of the endometrial tumors. The pathogenesis of such lesions following appendicectomy is a special problem, however, and we shall not attempt an explanation of this phenomenon in that small group other than to state that they are not quite entirely analogous to the postcesarean cases. The longest recorded interval between a cesarean section and the appearance of a cicatricial endometrial tumor is about ten years;[9] and, even in this case, it is quite likely that the lesion had been present many years before it came to the attention of the patient.

The nodule is generally about the size of a walnut, is quite hard in consistence, and causes little spontaneous pain. Only slight tenderness is present except during the menses, when there is generally a moderate swelling of the tumor, accompanied by severe pain and tenderness localized to the nodule. The concomitance of the pain and the menstrual period is almost a pathognomonic symptom and is found in nearly all the cases,[10] although exceptions to this rule do occur.

In Mengert's case[11] the pain was constantly present but was increased during the menses. Rarely, as in Neuweiler's case,[12] the pain bears no apparent relationship to the menses; while in von Franqué's[3] case the tumor produced no pain at all. In one of Harbitz'[13] cases (Case 13) the tumor regularly produced comenstrual pain, but this pain ceased entirely when the patient became pregnant, only to return when the regular menstrual cycle was restored after delivery.

Undoubtedly the pain is produced by the participation of the endometrial tumor in the corresponding cyclic changes of the normal endometrium lining the uterus, in response to hormonal stimulation. Such

changes produce a miniature menstruation in the tumor itself with consequent increase in tension and attendant pain. Such an hypothesis is supported by the observation of Heaney[4] and others, who noted that the skin overlying the tumor became bluish in color during the menses. In some cases, notably those of Stavely,[14] Orestano,[15] and Rieck,[16] the skin over the tumor broke down at such times with the periodic discharge of hemorrhagic fluid. Such an occurrence would lead one to suspect the presence of a uteroparietal fistula, but probing of the sinus or x-ray examination after the injection of an opaque medium into the sinus should serve to effect the differentiation. Rarely, as in Lützenkirchen's case,[17] the endometrial tumor itself communicates with the uterine cavity, in which instance the anatomic connection should be demonstrable at the time of the operative exploration.

Occasionally the nodule may be mistaken for a small ventral hernia containing incarcerated omentum, or for a desmoid tumor;[18] but a careful inquiry into the history and the periodicity of the pain should serve to permit a proper differential diagnosis.

PATHOLOGY

Endometrial tumors usually take the form of discrete well-localized nodules, although Williams[19] has reported an instance of a diffuse infiltration of the entire scar. The nodules are usually single, but cases in which two nodules have been found are recorded.[20, 21] There appears to be no predisposition to any particular localization in the upper or lower portions of the wound, and implants have been found on either side of the midline in cases where the transverse Pfannenstiel incision had been used.[22] Localization may take place in any or all of the layers of the abdominal wall comprising the laparotomy cicatrix, the most usual site being the superficial surface of the anterior rectus sheath. In Berkeley's case[23] the nodule involved only the skin and subcutaneous tissue. Harbitz[13] mentions one case (Case 9) in which the nodule was entirely in the subcutaneous tissues. In other reports[24, 25] all the layers of the abdominal wall were involved. As a rule the parietal peritoneum is not involved. Invasion of the intraperitoneal structures, while unusual, has been noted by several writers,[26, 27] the adherent structure generally being the fundus of the uterus. Such a finding does not necessarily mean that there is direct continuity of endometrial tissue from the uterus to the parietal implant, for microscopic examination may reveal the adhesions to be purely fibrous in character.[27]

The tumors are very firm and their consistence has aptly been compared by Lemon and Mahle[28] to that of a uterine fibromyoma. Section reveals the bulk of the tumor to be comprised of interlacing bands of white and gray fibrous tissue which penetrate beyond the ill-defined limits of the tumor into the adjacent areolar tissue, fascia, muscle, and

peritoneum. Irregular, softer areas between the fibrous bundles consist of glandular tissue and small cystic collections of old blood and mucoid material. In one case, Harbitz' Case 14, the cyst formation was so pronounced as to constitute the bulk of the tumor. Serial sections have demonstrated[29] that these apparently isolated islands of epithelial tissue actually communicate with one another.

Microscopically the epithelial element is found to consist of typical endometrial mucosal cells arranged in tubules and small cysts resembling pseudoglomeruli at times. All the stages of the menstrual cycle are observed in them, depending upon the time of the excision of the specimen in relation to the patient's own menstrual cycle. In one of Harbitz' cases (Case 4), where excision was performed one day before the onset of the regular menstrual period, the specimen exhibited the characteristic progestational proliferation one would expect to find in the normal premenstrual endometrium. This participation of the tumor in the cyclic changes of the normal endometrium is even more strikingly shown by the experience of Martin, Michon, and Pigeaud,[30] who excised an endometrial tumor of a laparotomy scar during a subsequent cesarean section at term, and who found, on histologic examination of the specimen, that the tumor cells exhibited the decidual reaction of pregnancy.

The nonepithelial elements of the tumor consist of a varying amount of connective tissue which may or may not be accompanied by smooth muscle fibers.[31]

PATHOGENESIS

Many ingenious hypotheses have been promulgated to explain the occurrence of ectopic endometrial tissue ever since this lesion was first observed. In 1896 von Recklinghausen[32] offered the theory that these tumors represented misplaced embryonic rests from the original Wolffian urogenital epithelium. Such an explanation, while conceivably applicable to cases of pelvic endometriosis, is obviously untenable in the postoperative cicatricial cases, for it has been established beyond controversy that the anterior abdominal wall is not involved by the Wolffian body at any stage of its development.

In 1908 Cullen[33] observed evidences of infiltration of the uterine wall by the basal layers of the endometrium in his cases of uterine endometriosis and concluded that direct invasion was the mode by which the endometrial epithelium migrated to other portions of the genitals. While such an hypothesis is fairly well supported in those cases in which the ectopic localization is limited to the immediate vicinity of the uterus, it becomes somewhat farfetched if one attempts to explain other instances of more distant implantation, such as have been commonly observed since Cullen's original reports. Certainly this theory could scarcely explain the occurrence of subcutaneous extraperitoneal nodules in the group of postcesarean cicatricial involvements.

In 1924 Halban[34] elaborated Cullen's hypothesis, and put forward a conception of lymphogenous "metastatic hystero-adenosis" as the explanation of more distant endometrial localizations. Such a theory, however, would presuppose the existence of unusual and improbable lymph channels from the uterus to the intestinal serosa, navel, etc. Furthermore, in the cases of endometriosis of the groin, it is not unusual to find the endometrial tumor adjacent to but not actually involving the inguinal lymph nodes. Here also it would appear that the hypothesis is not sufficiently inclusive to explain the formation of endometrial tumors in laparotomy scars.

More fundamental in its tenets than any of the preceding assumptions is the theory of serosa-epithelial metaplasia, subscribed to by numerous observers, notably Novak[35] in this country and Nicholson[36] in Great Britain. These investigators point out the close relationship of the germinal epithelium to the mesothelial celomic lining, both in the embryo and in the adult organism where the surface epithelium of the ovary is directly continuous with the peritoneum itself. This they consider as evidence of the potentiality inherent in all peritoneal mesothelium for the formation of true germinal epithelium under the influence of certain biological stimuli. Subscribers to this theory believe that the latent capacity of the peritoneal serosa for epithelial transformation is more or less directly proportional to its proximity to the line of normal serosa-epithelial transition (i.e., at the ovary). They point to the preponderance of involvement of the pelvic structures by endometriosis in support of this view and likewise indicate that even in the postcesarean laparotomy cases the endometrial tumors occur at a level below the umbilicus. Opponents of this theory reply, however, that such localization in wounds below the umbilicus is due merely to the fact that most incisions for gynecological or obstetrical operations are made below the umbilicus. This contention is supported by the observation of German,[21] who found two endometrial tumors in a postcesarean laparotomy scar, one of which was in the upper angle of the wound above the level of the umbilicus. The most serious objection to the theory of serosal metaplasia, however, is its failure to explain adequately the numerous instances of completely extraperitoneal localization of endometrial tumors in the abdominal laparotomy scars following cesarean sections and other gynecological operations.

A tremendous impetus to the study of endometriosis followed the publication in 1921 of Sampson's classical observations[37-39] and well-thought-out views on the pathogenesis of this lesion. He attributed ectopic localizations of endometrial tissue to the dissemination and implantation of adult, differentiated, viable bits of endometrium which become engrafted upon a suitable metastatic site, there to proliferate if nutritive and other conditions permit of its survival. By this theory the usual intraperitoneal forms of endometriosis are explained by the

extrusion of bits of viable endometrium from the fimbriated ends of the Fallopian tubes due to partially retrograde menstrual flow. He further supported his conception by successful tissue culture experiments, since confirmed by other investigators, by which he showed that portions of endometrium cast off during menstruation are viable.

Sampson's theory is particularly applicable to our own group of cases for it is easy to predicate that a microscopic fragment of endometrium detached from the uterus during the operation may become lodged in the tissues of the abdominal wound, and, under favorable conditions, continue to grow there as a tissue transplant. The actual detachment of the uterine mucosa may be accomplished by the scalpel or, as suggested by Lochrane,[40] by the passage of a needle into or through the endometrium during suturing. Additional evidence in favor of this theory is provided by Violet's[41] and Prager's[42] reports of extraperitoneal endometrial tumors of the perineum following deliveries from below in which wide tears into the perineal tissues occurred during labor.

In view of the fact that Sampson's implantation theory is the only one of the entire group which explains extraperitoneal as well as intraperitoneal localizations of endometriosis, we believe it should be accepted as the most valid comprehensive theory of the pathogenesis of this lesion in all locations.

PROPHYLAXIS

One practical application of the implantation theory in the prevention of cicatricial endometrial localization is a modification of the technique of hysterotomy for cesarean section or other purposes. Care should be taken to provide adequate protection of the abdominal wound margins by covering them with gauze pads or rubber tissue as suggested by Bonney.[43] Furthermore, it is recommended that, following the closure of the peritoneum, the wound be flooded with ether or absolute alcohol to remove not only loose and traumatized portions of fatty tissue but likewise to devitalize or remove mechanically any minute fragments of endometrium which might otherwise have been allowed to gain a nutritive foothold in the wound. Schwarz[44] advises that a low cervical incision be made where possible when performing a cesarean section. This suggestion is made on the presumption that the cervical mucosa thus exposed is less likely to become implanted in the abdominal wall. In this connection, however, it is interesting to note that in Rieck's[45] case the occurrence of an endometrial tumor in the scar followed a Latzko cesarean section.

TREATMENT

In most cases complete excision of the nodule or nodules is feasible and is advisable as the procedure of choice. Care should be taken to effect a wide dissection into normal surrounding tissues, because these

tumors do not have a well-defined capsule. Usually it is not necessary to open the peritoneum, but, unless the tumor is definitely not attached to the peritoneum at all, it is advisable to remove the underlying peritoneum along with the tumor. If local excision is impossible for technical reasons or because of the general condition of the patient, treatment should consist of the application of deep radiotherapy to the ovaries as is done in inoperable intraperitoneal endometriosis. By this means the menstrual cycle is halted and the endometrial tumor, though still present, does not produce the usual comenstrual pain.

SUMMARY

1. Attention is directed to a group of endometrial tumors occurring in postcesarean abdominal wall laparotomy scars.

2. We believe these tumors to represent proliferations of fragments of uterine mucosa inadvertently transplanted into the abdominal wound during a previous hysterotomy.

3. Clinical features, including the pathognomonic comenstrual pain, pathology, and pathogenesis are discussed.

4. Prophylaxis and treatment are outlined.

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THE ETIOLOGY OF VASOMOTOR AND NUTRITIONAL CHANGES FOLLOWING PERIPHERAL NERVE SECTION

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FOR many years surgeons have known that vasomotor and nutritional changes may occur in regions (particularly the digits) innervated by a peripheral nerve which has been sectioned. However, the cause of these phenomena has remained obscure. Following section of a peripheral nerve, the cutaneous area innervated by it becomes at once hot and dry. This initial period of vasodilatation lasts for only a relatively short period of time. It is followed by vasoconstriction, as indicated by a drop in skin temperature. Unless rapid regeneration of the divided nerve takes place, the denervated part becomes cold to palpation; color changes appear in the skin, atrophy of skin, subcutaneous tissue, and bone ensues; characteristic changes occur in the nails; and ulcers which fail to heal may appear. While some of these changes may be explained on the basis of disuse, lack of active function does not tell the entire story. Likewise, the theory of the "trophic" function of nerves is more specious than convincing. Perhaps through the application of certain newer anatomic and physiologic principles, an explanation of these phenomena may be forthcoming.

The initial phase of vasodilatation with loss of sweating presents a picture similar to that following sympathectomy. In fact, it is based on the severance of postganglionic sympathetic neurones. Kramer and Todd[1] were the first to show that in the upper extremities the postganglionic sympathetic fibers to the blood vessels are distributed within the branches of the brachial plexus. Potts,[2] working in Dr. Todd's laboratory, found the same condition obtaining in the inferior extremities; namely, that the postganglionic sympathetic neurones to the blood vessels are carried within the branches of the lumbosacral plexus. Up to this time it had been supposed that vasomotor fibers in the extremities were carried in the adventitia of the main arterial trunks and their branches, as they are in the visceral vessels. The aforementioned work coming from Dr. Todd's laboratory has been confirmed many times. Woolard and Phillips[3] demonstrated that the sympathetic nervous innervation of the subpapillary arterioles corresponds to the somatic cutaneous innervation. To those working in

Received for publication, June 6, 1938.

the field of peripheral circulatory disease, it is well known that blocking the ulnar, median, posterior tibial, or peroneal nerves normally produces a vasodilatation with loss of sweating in the cutaneous zones innervated by these nerves.

Recognition of the fact that surgical or traumatic division of a peripheral nerve will denervate the blood vessels in the regions innervated by that nerve gives us a clue to the etiology of the various vasomotor and nutritional disturbances which ensue. In 1934 Freeman, Smithwick, and White[4, 5] published the results of their classic experiments on the sensitization of blood vessels in the sympathectomized human extremity to circulating adrenaline. A detailed account of these experiments, together with their background and their practical application to the problem of sympathectomy, is given by White.[6] A brief exposition of their findings is sufficient for our thesis. Their experiments were performed on individuals suffering from Raynaud's disease, in whom the postganglionic sympathetic fibers to the blood vessels of the upper extremities had been destroyed by cervicothoracic sympathetic ganglionectomy. They found that, beginning around the seventh to the fourteenth postoperative day, an intravenous injection of a quantity of adrenaline insufficient to cause vasconstriction in normal regions would produce an intense arteriolar constriction in the fingers of the denervated extremity, as evidenced by decreases in their skin temperatures. These denervated arterioles were similarly hypersensitive to other vasoconstricting stimuli, as exposure to cold.

We became interested in determining whether similar sensitization would occur in normal arterioles following traumatic division of a peripheral nerve. Since arterioles thus sensitized may ultimately become permanently constricted with resultant loss of nutrition to the tissues they supply, the so-called trophic function of nerves, and the vasomotor and nutritional changes following their division, on this basis, could be explained. An opportunity to test this hypothesis was afforded us with the admission of a case of complete traumatic division of the median and partial division of the ulnar nerves at the left wrist.

CASE REPORT.—A. D., No. 187161, was admitted to Surgical Service A of Cleveland City Hospital with a deep laceration of the left wrist sustained in an auto accident. He was taken to the surgery at once, and the wound was explored. It was found that, in addition to other structures, the median nerve was completely severed and the ulnar nerve partially severed. The divided ends were united by suture.

The following day, he presented complete anesthesia of the palm and volar surfaces of all the digits, with the exception of a narrow strip at the outer border of the fifth finger and hypothenar eminence. The anesthetic zones were red, hot, and dry, exactly similar in appearance to a recently sympathectomized hand.

On the thirteenth day following the injury, skin temperature readings, stabilized at a room temperature of 24° C., were taken from the pads of the distal phalanges of the fingers of both hands. They were:

	RIGHT (CONTROL)	LEFT (ANESTHETIC)
First finger	32.2	33.2
Second finger	32.0	33.0
Third finger	31.7	33.0
Fourth finger	32.4	33.3
Fifth finger	32.1	33.5

It is seen that at this time the skin temperatures of the anesthetics fingers were higher.

Skin temperature readings, stabilized at a room temperature of 23.4° C., were taken from the same spots one week later and were:

	RIGHT (CONTROL)	LEFT (ANESTHETIC)
First finger	31.1	29.1
Second finger	31.1	28.6
Third finger	31.1	28.8
Fourth finger	31.8	28.6
Fifth finger	31.6	28.3

Evidently something had occurred in the intervening week, for now the skin temperatures of the anesthetic fingers were not only relatively lower, but were absolutely subnormal. At room temperatures above 22.5° C., stabilized skin temperatures of the fingers should normally be at least 31° C.[7]

Immediately following the taking of these readings, 0.35 c.c. of a 1 per cent solution of neosynephrine hydrochloride was injected subcutaneously in the left leg, and skin temperature readings from the same points were taken at five-minute intervals. Fortunately for the purposes of this experiment, the room temperature began to rise rapidly from 23.4 to 26.5° C. following the injection. Thus, a drop in the skin temperatures of the anesthetic finger pads in response to the injection would be very significant; and particularly so if, despite the injection, the control readings from the normal finger pads should rise in response to the increasing room temperature. The readings actually obtained were:

RIGHT (CONTROL)

FINGER	FIRST	SECOND	THIRD	FOURTH	FIFTH
RESTING TEMPERATURES	31.1	31.1	31.1	31.8	31.6
SUBCUTANEOUS INJECTION	0.35 c.c. 1% NEOSYNEPHRINE HYDROCHLORIDE LEFT LEG				
5 min. later	31.1	31.0	31.3	32.0	32.0
10 min. later	31.5	31.5	31.7	32.0	32.0
15 min. later	31.8	31.7	31.7	32.0	32.1
20 min. later	32.0	31.9	31.9	32.2	32.4
25 min. later	32.0	32.0	32.3	32.5	32.5
30 min. later	32.3	32.2	32.5	32.5	32.5

LEFT (ANESTHETIC)

FINGER	FIRST	SECOND	THIRD	FOURTH	FIFTH
RESTING TEMPERATURES	29.1	28.6	28.8	28.6	28.3
SUBCUTANEOUS INJECTION	0.35 c.c. 1% NEOSYNEPHRINE HYDROCHLORIDE LEFT LEG				
5 min. later	28.8	28.5	28.3	28.1	28.1
10 min. later	28.2	27.7	27.5	27.3	27.1
15 min. later	27.8	27.5	27.2	27.0	27.0
20 min. later	27.4	27.1	26.7	26.6	26.7
25 min. later	27.1	26.7	26.4	26.4	26.4
30 min. later	26.6	26.2	26.1	25.9	26.1

It is seen that, whereas the control temperatures rose, those from the anesthetic finger pads decreased, the drop reaching its maximum 30 minutes from the time of injection of the vasoconstricting drug.

The phenomenon of sensitization of normal human arteriolar musculature to a vasoconstricting stimulus is clearly demonstrated. That such sensitization to vasoconstricting influences could be the etiology of vasomotor and nutritional changes in denervated tissues is certainly possible. Unfortunately, there is no way of preventing the onset of this sensitization. However, it should be possible to keep its deleterious effects at a minimum until regeneration of the divided nerve has taken place. The denervated part should be kept warm at all times, emotionally upsetting situations should be avoided, and the use of tobacco interdicted. In addition, various measures designed to produce a regional vasodilatation should be employed, such as hot paraffin baths, massage, diathermy, and mecholyl iontophoresis.

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A NEW MODIFICATION OF SUBARACHNOID ALCOHOL INJECTION FOR THE BILATERAL BLOCKING OF THE LOWER SACRAL NERVES IN INTRACTABLE PAIN OF THE PELVIC VISCERA

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THE intrathecal injection of alcohol to block the posterior sensory roots of the spinal nerves was proposed by Dogliotti[1, 2] as a substitute for posterior rhizotomy or section of the spinothalamic tract. During the last seven years, this method has come into fairly widespread use, and reports of numerous successful results have been published.[3-6] Radical as this method at first seemed, it is now generally realized that it carries but little greater risk of injury to the motor or bladder pathways than does cordotomy; in addition, it can be used in patients with hopelessly advanced malignant disease who could stand no open operation. The chief objections to subarachnoid alcohol injection are its narrow margin of safety and the fact that doses of less than 1 c.c. of absolute alcohol so often fail to produce adequate or lasting analgesia. The limitations of the method have been brought out very clearly by the neuropathologic studies of Aird and Naffziger.[7] When two injections have to be made for bilateral pain, the risks of failure or complication are considerable.

A modification of Dogliotti's method of subarachnoid alcohol injection has been developed in the Neurosurgical Clinic of the Massachusetts General Hospital which has given uniformly successful analgesia over the sensory distribution of the lower sacral nerves on both sides* (Fig. 1). Pain limited to the penis, the posterior half of the scrotum or labia, the perineum and saddle area in the buttocks is seen not infrequently in inoperable cancer and certain other diseases of the bladder, prostate, and lower rectum. It can be a particularly distressing type of pain which remains localized to this area as long as the disease is confined to the neighboring soft tissues and has not spread to bone or involved the upper trunks of the lumbosacral plexus. Although the method has been in satisfactory use for over two years, it seemed best not to report it until it had been subjected to a sufficient number of trials to demonstrate that it could be counted on to give consistent results and to be free from serious risk.

Received for publication, June 9, 1938.

*Adson,[5] in a report of 40 cases treated by Dogliotti's intrathecal alcohol injection at the Mayo Clinic, has mentioned the possibility of blocking the lowest sacral nerves bilaterally. He has used a somewhat similar technique in two cases of anal and vulval pruritus.

As in Dogliotti's technique for blocking posterior nerve roots on one side, bilateral infiltration of the roots of the lower three sacral nerves in the caudal end of the subarachnoid space depends on the fact that absolute alcohol (specific gravity 0.775 at body temperature) is lighter than spinal fluid (specific gravity 1.005). However, instead of making the injection in the lateral position, the patient is laid prone and the sacrum elevated by lowering the two ends of the operating table. The lower termination of the subarachnoid space then becomes the highest

Fig. 1.—Area of anesthesia following bilateral alcohol injection of lowest sacral nerves (Patient 4). Solid black indicates area of lasting analgesia. The larger area down the posterior thighs which is enclosed by black lines indicates outlying zone of hypoesthesia immediately after injection. This disappeared within a few hours.

point in the cerebrospinal fluid column. (Fig. 2.) Lumbar puncture can usually be carried out in this position, either between L4 and L5 or between L5 and S1. When the needle enters the subarachnoid space, spinal fluid must be aspirated with a syringe, because it usually is not under sufficient pressure to flow out spontaneously. Only a minimal amount of fluid should be withdrawn to check the position of the needle. One to 1.2 c.c. of sterile absolute alcohol* should then be injected. The

*The use of sterilized alcohol is recommended for this purpose, as certain spore-forming microorganisms may remain viable even in absolute alcohol. This can be obtained in ampoules from the Intravenous Products Company of North America, 239 Fourth Avenue, New York, N. Y.

alcohol floats upwards to the termination of the subarachnoid space and bathes the caudal ends of the third, fourth, and fifth pairs of sacral nerve roots. The injection produces no sensation beyond a vague feeling of warmth, but causes rapid anesthesia of the buttocks over a radius of about three to six inches around the anus, a little way down the posterior aspect of the thighs, and forward over the perineum, posterior scrotum, and penis (Fig. 1). In order to allow the alcohol to become fixed in this area, it is necessary to keep the patient perfectly quiet and in the same position for over an hour. A moderate dose of morphine is helpful to prevent discomfort and restlessness. After the patient has been re-

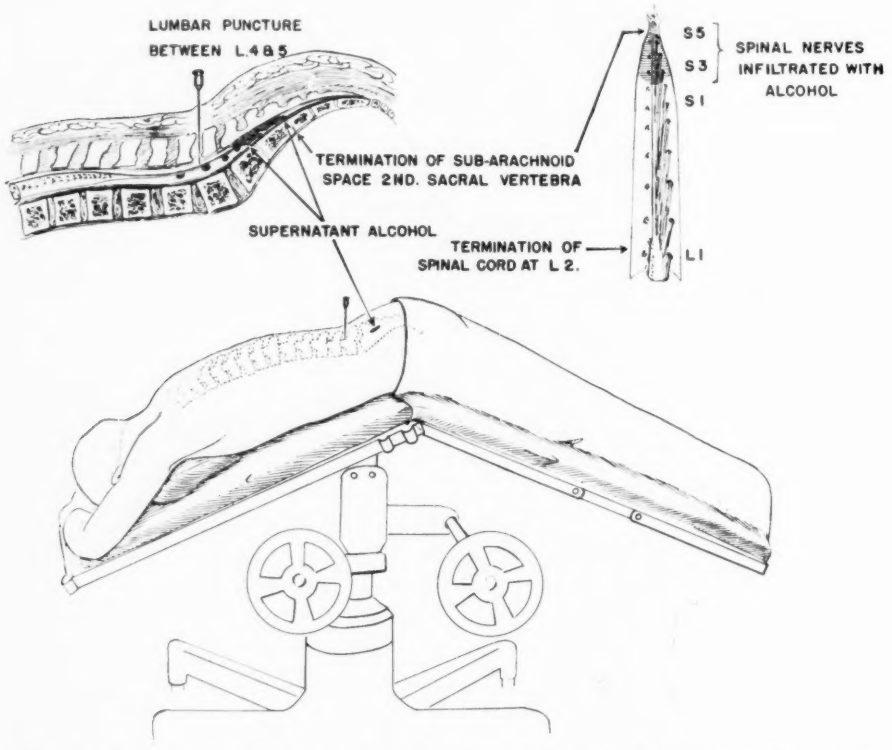

Fig. 2.—Technique of bilateral subarachnoid alcohol block of lower sacral nerves.

turned to bed, it is only necessary to insist on the routine flat in bed position in order to prevent headache, which is liable to follow any lumbar puncture if the head is raised too soon. The patient can ordinarily be up and walking around the next day, as though nothing more than a diagnostic puncture had been performed.

Table I contains a brief summary of the case histories, past treatment, and the results of subarachnoid alcohol injection of the lower sacral nerves in six patients. The loss of cutaneous sensation has been confined to the distribution of the three lowest pairs of sacral nerves. While there was immediate relief of pain in every instance, the duration of re-

TABLE I

PATIENT	DIAGNOSIS	PREVIOUS OPERATION	SYMPTOMS	ALCOHOL INJECTION	COURSE
1 Male, 66	Carcinoma of rectum, recurrent in perineal scar	Combined abdomino-perineal resection 1 yr. previously	Good result for 9 mo., then reopening of perineal wound with bloody discharge and deep perineal pain; liver enlarged and nodular	4/9/36: 1.2 c.c. 95% alcohol	Complete relief for 2 wk., then recurrence; unfortunately he lived too far away to return for reinjection; no disturbance in urination
2 Male, 69	Carcinoma of rectum	Colostomy 2½ yr. previously	Functioning colostomy; deep perineal pain and large, nodular liver	4/17/36: 1.2 c.c. 95% alcohol	Local doctor wrote that, as far as he could judge, the patient "did not suffer any great amount of pain"; there was no disturbance in urination, but he was able to retain his rectal discharge less well. Died 6 mo.
3 Male, 51	Carcinoma of bladder	3/23/36: Resection and electrocoagulation 10/23/36: Transurethral fulguration Suprapubic tube	Severe recurrent pain from sacrum to penis with hematuria; dehydrated and in poor condition	11/16/36: 1 c.c. 95% alcohol	Severe sacral and penile pain entirely relieved. Died 11 days later of terminal pneumonia
4 Male, 69	Carcinoma of prostate	9/22/36: Suprapubic cystostomy and permanent drainage	Patient bedridden and unable to rest on account of painful spasms in perineum and penis	1/8/37: 1.1 c.c. absolute alcohol	Complete relief of pain for 15 mo.; his rectum remains insensitive, so that he occasionally soils himself when his bowels are loose; at 20 mo., there is a slight recurrence of his old pain, but he does not require opiates; bladder full of cancer
5 Male, 68	Carcinoma of bladder	3/26/37: Bladder fulguration and implantation of radium seeds; suprapubic cystostomy Suprapubic tube	Bad pain on urination which radiates from sacrum through perineum to penis; much bladder irritability and frequency	3/27/37: 1 c.c. 95% alcohol 6/4/37: 1.1 c.c. 95% alcohol	Complete relief for a month, then a gradual recurrence, for which injection was repeated; discharged relieved on following day; at 11 mo. he complains of some suprapubic discomfort, but his old perineal pain seems to remain relieved
6 Male, 67	Carcinoma of prostate	2/23/37: Vasectomy and permanent suprapubic cystostomy	Excruciating pain at base of penis on every movement	4/10/37: 1.1 c.c. absolute alcohol	Complete relief of perineal pain with definite hypoasthesia for 10 mo.; at end of a year he is beginning to have a slight recurrence, but can still get along without morphine; at first there was some relaxation of anal sphincter, but no leakage unless he had diarrhea

lief has been somewhat variable. In Patient 4, who has been followed over the longest period, complete freedom from pain lasted for fifteen months, and at twenty months he still does not require opiates. If the sensory fibers recover, reinjection is a simple matter. Patients 1 and 5 had a gradual recurrence of their former pain. The latter was treated successfully by reinjection, but the former lived at such a distance that it was impossible for him to return to the hospital on account of his poor general condition.

Complications in these particular patients have been minimal. None have noted any weakness of their legs. As a rule the injection produces some relaxation of the anal sphincter and an insensitive lower rectum, so that patients may soil themselves when their bowels are loose. None, however, have complained of frequent fecal incontinence. As far as bladder function is concerned, the two patients with rectal cancer who were not already on constant drainage observed no impairment in their ability to urinate, although it seems too optimistic to believe that this will consistently be the case.

There is a valid anatomical explanation for these clinical observations. In spite of the fact that in the lower cauda equina the sensory and motor roots lie so close together that some of the injected alcohol is certain to come in contact with the latter, it would be most unlikely for the motor fibers to the legs to be involved, because the action of the alcohol is so accurately confined to the lowest sacral roots below the origins of the sciatic nerve. In addition the observations of Gasser and Erlanger[8] have shown that the large heavily myelinated axones to skeletal muscle are far less vulnerable to the action of drugs, such as procaine and alcohol, than the thinly myelinated or unmyelinated pain fibers. Inasmuch as the parasympathetic axones which conduct the principal impulses mediating urination and defecation are unmyelinated, it may seem surprising that these functions are not more severely impaired. Anatomical studies of Simmons[9] have traced the bladder outflow to the second and third sacral roots but have shown that the innervation of the lower rectal segment and the anal sphincters is given off one segment lower. This is probably the explanation why three of these patients noted a slight disturbance in bowel control.

In spite of the fact that bladder paralysis may be infrequent, subarachnoid alcohol injection undoubtedly carries a greater risk of this complication than bilateral cordotomy. It should therefore not be recommended for any patient who is not already on constant drainage or is not prepared to accept it. Inasmuch as the majority of patients with intractable pain in this area are suffering from cancer of the prostate or bladder neck, the necessity for an inlying catheter or suprapubic cystostomy has usually been reached. Injection was performed in the two patients with rectal carcinoma because their poor general con-

dition rendered cordotomy out of the question, but the intensity of their pain made them quite willing to accept the possibility of a catheter existence.

In conclusion, subarachnoid alcohol injection of the lower sacral nerves is not intended to supplant cordotomy in patients who are favorable operative risks, but for those who are in too poor condition and have only a short time to live, it is a most valuable substitute.

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ACUTE AEROBIC (NONPUTRID) ABSCESS OF THE LUNG

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A. INTRODUCTION

THE most serious pulmonary complication of so-called "acute suppurative bronchopneumonia," is nonputrid (aerobic) pulmonary abscess. Until recent years we considered the term "abscess of the lung" to be practically synonymous, from the clinical standpoint, with putrid (anaerobic) abscess, and regarded the nonputrid (aerobic) varieties to be rare and of clinical importance only when complicated by infection of the pleura.[1] As a result of a great increase in the incidence of "suppurative bronchopneumonia" in the past few years, however, and its recognition as a clinical entity, we have encountered a substantial and possibly disproportionate increase in the number of cases of aerobic abscess. Thus we have altered completely our original views and now regard nonputrid abscess of the lung as a not uncommon disease of considerable clinical significance. It is also a disease for which, in our opinion, surgical consideration and operative treatment are required under certain circumstances. Because we have not found an adequate clinical survey of the subject in the literature, we shall present it in some detail. Inasmuch as this paper is based on personal observations and experiences, surgical aspects will of necessity be stressed, and operative treatment will receive special consideration.

B. THE CAUSATIVE BRONCHOPNEUMONIA

In order to deal logically with the subject of nonputrid pulmonary abscess, the characteristics of the causative bronchopneumonia should be noted. We shall proceed, therefore, with a brief discussion of some basic features. Pathologically, there are two prominent characteristics of the bronchopneumonia from which a nonputrid pulmonary abscess is derived, namely *suppuration* and *necrosis*. The last appears to be an essential and ofttimes outstanding feature of the pathologic process. The term "suppurative bronchopneumonia," which is in common use, does not emphasize this feature (necrosis) and therefore seems inadequate. Accordingly, we have devised and shall employ the more descriptive term "necrosuppurative bronchopneumonia" which denotes the coexistence of both characteristics. In the classification which we have drawn up, "necrosuppurative parenchymitis" has been placed

Received for publication June 27, 1938.

in a separate category. The reason becomes apparent upon an inspection of the table, and the features of the lesion will be referred to briefly in a succeeding paragraph. The identification of the various pathologic processes mentioned in the classification is based on personal observations. The latter consist partly of a study of roentgen features and operative findings, and partly of observations of clinical features and of autopsies. Unfortunately, a comparison with the observations of others cannot be made because of the paucity of literature on the subject. The classification which we have drawn up does not purport to be all inclusive, its particular purpose being to emphasize the surgical aspects of the disease under discussion. Arbitrary lines have been drawn and undoubtedly result in sharper cleavage between lesions than is actually the case. However, the classification epitomizes the observations which we have made.

A CLASSIFICATION OF ACUTE PULMONARY SUPPURATION DUE TO AEROBES, WITH SPECIAL REFERENCE TO SURGICAL ASPECTS

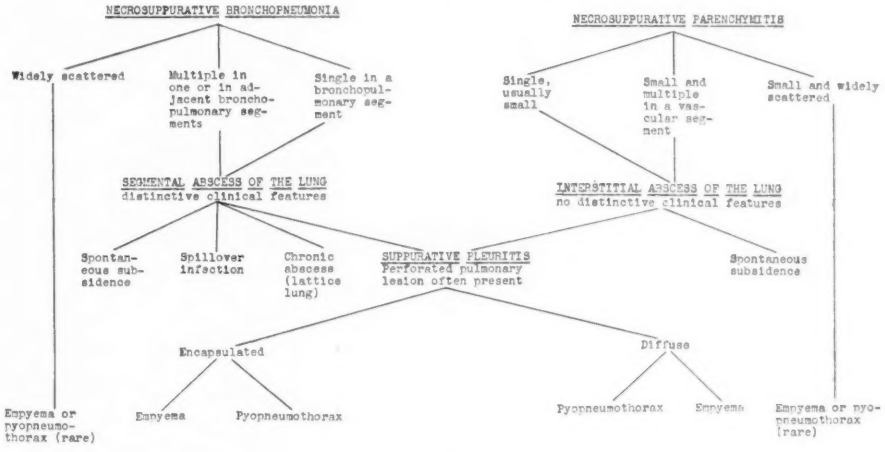

Fig. 1.

The features of necrosuppurative bronchopneumonia now will be outlined. The disease is characterized: (1) pathologically, by the coexistence of suppuration and necrosis involving one or more bronchopulmonary segments;* (2) clinically, by the occurrence of fever and cough, and the expectoration of odorless pus; (3) roentgenologically, by the presence of one or more areas of pneumonic infiltration; (4) bacteriologically, by the presence of aerobic, pus-producing bacteria in the sputum.

Although amplification of this bare outline is desirable, lack of space does not permit of a discussion of the variations which have been noted. Fever is often high, may be remittent, and is attended

*The term "bronchopulmonary segment" has been designated by Glass (Am. J. Roentgenol. 31: 1934) as a secondary bronchus with its tributary bronchioles and parenchyma. Each segment is separate and distinct from adjacent ones.

frequently by profuse sweats. The sputum is frankly purulent and often copious. It is usually of greenish or grayish tinge and is either entirely free from odor or has a faintly musty smell. Severe pain in the chest at the onset and hemoptysis when the expectoration of pus begins, are occasional features. These are not present uniformly, as in the case of putrid pulmonary abscess. The degree of dyspnea and of prostration varies considerably.

The characteristic roentgen film in cases of necrosuppurative bronchopneumonia discloses an area of pneumonic infiltration involving a substantial portion of a lobe, the density of the shadow usually being greater than that seen in the nonsuppurative type of bronchopneumonia. The film may disclose a single lesion, multiple (discrete or coalescing) lesions in one lobe, involvement of more than one lobe, bilaterally situated lesions, or other bizarre combinations. The pathognomonic roentgen sign of necrosuppurative bronchopneumonia is rarefaction within the area of pneumonic infiltration. This may occur in the form of one or more circular areas of diminished density, as a single zone or as multiple ill-defined lighter zones situated within the area of consolidation. It is not seen in all cases and differences of opinion arise, not infrequently, in the interpretation of questionable areas of rarefaction. When a fluid level is seen, the formation of an abscess may be assumed to have begun.

The physical signs are essentially those of nonsuppurative bronchopneumonia, but the percussion note over the area or areas of dullness is apt to be more flat. At times, areas of involvement disclosed by x-ray examination may not be detected by the customary physical examination.

The clinical course of necrosuppurative bronchopneumonia is not predictable. It ranges from a short-lived infection ending in early recovery to a fatal form characterized by prolonged fever, profuse purulent expectoration, and spread of the pulmonary infection.

It is evident from all of the foregoing that the stage at which necrosuppurative bronchopneumonia becomes pulmonary abscess cannot be defined precisely. Thus, on the basis of a number of observations, we note that apparent transformation may take place rapidly or gradually and may occur at any period in the course of the disease. A transitional stage undoubtedly exists, but, since it cannot be identified by any available diagnostic method, it will not be included in the consideration of pulmonary abscess which now follows.

C. AEROBIC PULMONARY ABSCESS

Aerobic abscess of the lung occurs in two forms, "interstitial" and "segmental." Interstitial abscess has been discussed in another communication.[2] Again we point out, however, that interstitial abscesses

(1) are usually much smaller than the segmental variety, (2) usually are multiple, (3) may lie superficially or deeply in the lung, (4) are of varied etiology, and (5) ordinarily present no clinical (and no surgical) features unless or until the pleura is invaded.

The second type of aerobic pulmonary abscess is, by contrast, a lesion of substantial proportions. It occupies much or all of the bronchopulmonary segment which had been involved in the antecedent necrosuppurative bronchopneumonic process, and we therefore term the lesion a "segmental" abscess of the lung. It is with this type of lesion alone that we are concerned henceforth. The subject of acute segmental pulmonary abscess will be taken up from the standpoints of pathology, symptomatology, physical signs, diagnosis, and therapy.

I. *Pathology.*—From the viewpoint of pathology, there are a number of features which resemble closely and some which are quite dissimilar from those of acute putrid abscess of the lung. Perhaps the most striking difference lies in the strict localization of a putrid abscess to one part of the lung in the great majority of cases; whereas, a nonputrid abscess may exist in one bronchopulmonary segment, simultaneously with areas of necrosuppurative bronchopneumonia elsewhere in the lung. In other cases, multiple nonputrid abscesses may be present in several parts of the lung on one side, or on both sides, and at the same time areas of necrosuppurative bronchopneumonia exist in still other parts. Another contrast is to be seen in the extent of the infection of the parenchyma about the abscess. A putrid abscess of the lung begins as a localized area of gangrenous bronchopneumonia and the lesion, by the time it is recognizable clinically, usually is a well-defined abscess surrounded by a limited area of pneumonitis. By contrast, the nonputrid abscess throughout most of the clinical course may be, and often is, situated in the midst of extensive necrosuppurative bronchopneumonia. Thus, despite close resemblance between the two varieties in some instances, each presents distinctive features which warrant separate classification and consideration.

The statements to be made in regard to the gross pathology of nonputrid pulmonary abscess are based largely on observations made at operation combined with studies of roentgen features. We have designated as pulmonary abscesses all lesions in which collections of pus of substantial proportions existed within the substance of the lung, regardless of the presence or absence of adjacent bronchopneumonia. We also have termed "acute" all pulmonary abscesses of less than six weeks' duration.

The interpretation of the pathology of scattered pulmonary abscesses is based essentially upon roentgen films and occasional autopsies, for patients with such lesions rarely have been subjected to surgical treatment. These lesions are similar, pathologically, with soli-

tary pulmonary abscess which will be considered directly. However, the abscesses usually are small (although occasionally they attain large size), and the enveloping infiltration generally is rather limited. Invasion of the pleura is rare, and when it occurs the resulting pyopneumothorax almost invariably is encapsulated. Except when a complicating pleural infection exists, the scattered variety of aerobic pulmonary abscess is not a surgical lesion and will receive no further consideration.

There are at least three varieties of "segmental" acute aerobic abscess which, from a surgical viewpoint, merit consideration: (1) pulmonary abscess in the midst of, and apparently an incidental part of, an extensive necrosuppurative bronchopneumonia; (2) pulmonary abscess as the prominent or predominating lesion, but surrounded by a considerable area of necrosuppurative bronchopneumonia; (3) typical pulmonary abscess with a more or less sharply limited and narrow surrounding zone of infected lung.

Certain features are common to all three varieties. As already stated, the abscess is of substantial proportions, the range in diameter being from two to four inches or even more. The collection of pus is essentially monolocular, although recesses may be present. An important feature (as noted in all operative cases) is the extension of the abscess to, or almost to, the surface of the lung. This is to be anticipated, since a bronchopulmonary segment is involved. Overlying adhesions, agglutinating the apposed visceral and parietal pleurae, were found at operation in all cases. They usually were more edematous and less dense than in putrid pulmonary abscess, but sufficed in each instance for safe operative entry of the abscess in one stage. In the typical case the shell of lung between the abscess and the sealed-off pleurae is thin and practically avascular; in other instances, it is thicker and more vascular. The contents of the abscess vary from a considerable amount of pus under tension to air coexisting with relatively small quantities of pus. In cases of widespread excavation the outstanding characteristic is extensive destruction of bronchi. As a result, many bronchial orifices of varied size are to be seen at widely scattered points in the wall of the abscess. They generally are situated in recesses between fleshy ridges that rise from the floor and walls of the cavity. In other words, the interior of the cavity often presents the typical picture of fully developed "lattice lung" even when the lesion, from the viewpoint of duration, is as yet only in the acute stage. This picture is to be contrasted with the few bronchial orifices often existing at operation in cases of acute putrid abscess of the lung.

The surgical pathology of pulmonary abscess which perforates into the pleural cavity is worthy of special emphasis because of thera-

peutic implications. We do not refer here to the small cortical type (interstitial abscess) to which reference already has been made, but to "segmental" abscess. In the latter, expectoration of pus continues after rupture into the pleura has occurred, if the perforation does not permit of complete evacuation of the pulmonary abscess into the pleural space. Furthermore, the perforation may take place near the uppermost portion of the abscess and thus drainage into the pleura may be inadequate. The following three features of the pathology of perforation are of special surgical significance: (1) The perforation may be of insignificant size and covered by slough. (2) It may be so situated as to be difficult of detection; that is, in the re-entrant angle between visceral and parietal pleura, or on a ridge separating two unequal parts of a bilocular empyema, etc. (3) There may be two or more widely separated perforations resulting in two or more non-communicating empyemas.

II. *Clinical Manifestations.*—The symptomatology and physical signs of pulmonary abscess do not require extended discussion for they present few, if any, distinctive features. In general, the symptoms are indistinguishable from those of the pre- or coexisting bronchopneumonia. Occasionally, purulent expectoration may increase in amount, and fever may assume a more distinctly remittent character coincident with the development of a more definitely septic state. The physical signs of bronchopneumonia (q.s.) persist, rarely with the addition of signs of cavitation. The symptoms and physical signs of perforation into the pleura, with the formation of empyema, usually are not distinctive.

III. *Roentgenographic Features.*—Because of the paucity of physical signs and symptoms, the diagnosis of aerobic abscess of the lung must be based essentially on roentgenographic features. As already pointed out, in necrosuppurative bronchopneumonia a stage may be reached in which a number of more or less distinct areas of rarefaction, seen in the film, become well-defined cavities containing fluid levels. At this stage, localization of the suppurative process can be assumed to have occurred and an abscess of the lung therefore can be said to exist. In typical cases the fluid level becomes longer and the area of rarefaction (pulmonary excavation) larger. Multiple fluid levels not infrequently are seen within the zone of pulmonary infiltration. In other cases, the pulmonary cavity appears empty except for a small fluid level at its most dependent portion. A fluid level, pathognomonic of pulmonary abscess, was present at some stage of the disease in all of our cases in which the diagnosis was verified by operation. This is in contrast with the findings in certain cases of putrid pulmonary abscess in which no fluid levels were visible in the films.

An effort should be made to distinguish between the fluid levels of pulmonary abscess and those of pyopneumothorax. The extent of the fluid levels is of little value for this purpose, although the larger levels are more likely to be those of pyopneumothorax. On occasions, intra- and extrapulmonary fluid levels coexist in one film, and under such circumstances the difference between them is more readily recognizable. There are two roentgen features which characterize, and usually are to be noted in, the presence of pyopneumothorax. First, at fluoroscopic examination, the fluid level usually is much more mobile than that of pulmonary abscess. Secondly, in the postero-anterior view, the fluid level often extends to the thoracic cage, there being no lung intervening. Errors in diagnosis may occur, however, in spite of every effort to differentiate between perforated and unperforated pulmonary abscess. In our experience the error usually has been to make the diagnosis of pyopneumothorax while the lesion was still situated entirely within the substance of the lung.

In addition to the occurrence of a fluid level on the film, the change from bronchopneumonia to pulmonary abscess often is to be recognized by alterations within the area of pulmonary infiltration. As already pointed out, the infiltration due to bronchopneumonia is seen as a dense more or less homogeneous shadow. It is appreciably less dense and more broken up (mottled) in pulmonary abscess. When widespread pulmonary excavation occurs, much of the shadow is replaced by a single large area of rarefaction.

The discussion of the roentgen features of aerobic pulmonary abscess cannot be concluded without calling attention to the fact that, even when the pulmonary abscess is fully developed, one or more areas of bronchopneumonia may exist elsewhere in the lung. This fact is stressed because we, like others, occasionally have overlooked a small but tell-tale bronchopneumonic patch as a result of focusing attention on the obvious lesion.

IV. *Differential Diagnosis.*—A detailed analysis of the differential diagnosis of aerobic pulmonary abscess and other lesions which may simulate it would lead too far afield. However, brief reference should be made to several diseases which present similar features. Aerobic abscess is perhaps most often confused with pulmonary tuberculosis, since the resemblance between the two, both in clinical course and in roentgenological aspects, may be very close. Indeed, one of our first operative cases of aerobic pulmonary abscess was regarded for a time as a case of pulmonary tuberculosis. When doubt exists, the diagnosis of pulmonary abscess should be based upon the continued absence of tubercle bacilli from the sputum together with the persistence of profuse purulent expectoration. The differentiation should not be

difficult if films made at intervals over a period of several weeks are available. In occasional instances, bronchoscopy may be required. The latter is necessary more often, however, in order to distinguish between pulmonary abscess and pulmonary suppuration secondary to bronchial neoplasm. A number of cases have come under our observation in which the latter diagnosis was entertained because of cough, fever, emaciation, clubbed fingers, and a roentgen film disclosing dense pulmonary infiltration. Bronchoscopic examination established the diagnosis in these cases without difficulty. It also has proved invaluable in differentiating between putrid and nonputrid pulmonary abscess. As we have pointed out elsewhere,[3] putrid abscess is not always characterized by foul odor of the sputum. When the bronchus communicating with such an abscess is shut off by edema, expectoration may be purulent because of the associated bronchitis, and yet be completely free of odor. In addition, the roentgen films of putrid and nonputrid abscess may be identical. Under the foregoing circumstances, the two diseases may be indistinguishable until foul odor or foul pus is detected at bronchoscopy. In children the diagnosis of aerobic pulmonary abscess offers no difficulty, except in cases requiring differentiation from putrid abscess or from aspirated foreign body with secondary infection. Under such circumstances, bronchoscopic examination usually will establish the correct diagnosis.

V. *Preoperative Localization.*—We believe that accurate localization of the pulmonary abscess is the cornerstone of correct operative treatment, and some remarks on this subject are therefore in order. Since percussion and auscultation do not locate the lesion with precision, accurate localization is practically synonymous with roentgenographic localization. The roentgen films must be studied with the particular object of determining the site at which the pulmonary abscess is situated closest to the surface of the lung. (As has already been pointed out, agglutinating adhesions between the visceral and parietal pleura exist in that area.) Posteroanterior, lateral, and, at times, oblique pictures should be taken for this purpose, and fluoroscopy also is to be employed. With rare exceptions, bedside films are of very limited value for precise localization. The most favorable and perhaps the only site for safe surgical approach to the pulmonary abscess is a point directly over the fluid level. Here the abscess can be assumed to be nearest the visceral pleura. Ribs should be counted therefore with painstaking care, in order to be sure that the correct one will be resected at the time of operation. One should attempt also to determine with care the site at which the segment is to be removed from the chosen rib. If radio-opaque skin markers are employed during roentgenography, due allowance should be made for the shift of the overlying soft parts when the arm is placed in the appropriate position on the operating table. In short, every effort should be made to deter-

mine the precise situation of the most superficial portion of the pulmonary abscess, in order to approach the lesion safely through agglutinating visceroparietal pleural adhesions.

VI. *Operative Indications.*—The problem of operative indications now will be discussed. It should be stated at once that we are still in doubt in regard to a number of aspects of this question. In comparison with acute putrid abscess of the lung, the indications for operation in nonputrid (aerobic) abscess are less precise and clear-cut. The reasons are fairly obvious. A putrid abscess is a well encapsulated lesion; a nonputrid abscess not infrequently consists of a collection of pus in the midst of an active bronchopneumonia. Because of this difference, abrupt cessation of symptoms is the rule after operation for putrid abscess; whereas, little, if any, immediate result is apt to be noted after operation in some cases of nonputrid abscess (see case reports). Another reason for uncertainty in formulating the operative indications in cases of aerobic abscess is the ever present possibility of spontaneous recession of even large and extensively excavated lesions, particularly in children. Thus, a more conservative attitude toward operative treatment for nonputrid abscess than for putrid abscess is warranted.

The three forms of aerobic suppuration which were described in a preceding paragraph should be restated at this point, because they have a direct bearing on operative indications: (1) unencapsulated pus in the midst of necrosuppurative bronchopneumonia; (2) more or less circumscribed pulmonary abscess in the midst of necrosuppurative bronchopneumonia; (3) true encapsulated abscess. The last comprises the sole lesion for which operative treatment may be indicated. A source of difficulty, in our experience up to the present time, is the fact that this form is not always to be distinguished from the first and more particularly from the second form of suppuration. A true pulmonary abscess can be recognized with ease when the roentgen film is typical, for there exists a cavity with fluid level which is well demarcated from the adjacent lung by a narrow rim of infiltration. Unfortunately, this surgically remediable variety may be roentgenologically indistinguishable from the others when the cavity is more or less full and coexisting dense pleural reaction obscures the pulmonary lesion. Thus, we have operated in error in a few instances in which pus in the midst of bronchopneumonia and not a true pulmonary abscess existed. Although operation had no significantly untoward effect in these cases, the latter should not be subjected to operation if they can be recognized.

An important operative indication in pulmonary abscess is the imminence of or the actual presence of perforation into the pleura. In cases of obvious pleural invasion, the diagnosis usually can be made on the basis of physical signs and especially of roentgen film. How-

ever, the diagnosis of empyema may prove to be incorrect even when the percussion note is flat, the breath sounds distant, and a dense homogeneous shadow is present in the film. Indeed, the diagnosis may be incorrect even when aspiration reveals pus. Thus, we have operated for supposed empyema several times, only to find collections of pus confined to the lung. All proved to be cases in which multiple collections of pus existed in the midst of extensive bronchopneumonia, and accordingly operation was without appreciable beneficial effect.

In general there are three indications for operation in true pulmonary abscess: (1) to terminate a septic course which may prove fatal, as a result of further extension of suppuration or of spillover to other parts of the lung; (2) to prevent the development of empyema, when perforation of the lesion into the pleura seems imminent; (3) to drain the pleural collection, and to establish better drainage of a perforated lung abscess in cases of pyopneumothorax when the perforation is small. An indication for imperative operation seems to exist in all three of the above groups. Strictly speaking, however, it exists only in the third group and even then only insofar as the pleural infection is concerned. Spontaneous recovery might have ensued in some of the cases in the first group upon which we have operated. The following case is cited as an example of an entirely unexpected spontaneous recovery and illustrates that the clinical course may change with dramatic suddenness in this surprising disease.

W. K., Queens General Hospital, No. 32,274, a male, 39 years old, had been acutely ill for a period of three weeks suffering from pain in the right chest, high fever, and profuse purulent expectoration of pus. On admission to the hospital, extensive infiltration within the right lower lobe was demonstrated by roentgenography. At the end of two months, a large abscess developed in the right lower lobe. When an additional abscess, with fluid level, appeared in an adjacent part of the lobe and the patient's condition grew worse, surgical consultation was requested. The impression gained was that one or both of the abscesses already had perforated into the pleura. A large transfusion was given preparatory to operation. A roentgenogram taken on the day before that of the proposed operation revealed almost complete disappearance of the fluid levels and, accordingly, operation was deferred. The pulmonary lesions cleared rapidly thereafter. Follow-up roentgenograms demonstrate complete disappearance of pulmonary infiltration and the patient has remained well.

A strong argument in favor of operative treatment of true acute aerobic pulmonary abscess is the uniformly satisfactory outcome of operation. Thus, there has been no operative mortality, little postoperative morbidity, and complete cure in all such cases.

We are in doubt, at the present time, as to the procedure to be followed when a pulmonary abscess and one or more areas of bronchopneumonia coexist. The development of bronchopneumonia at a distance from the pulmonary abscess has been observed in several of our

cases. Such areas of bronchopneumonia may be assumed to be of spillover origin. Accordingly, their very presence may be considered an argument in favor of operation for the purpose of avoiding additional spillover lesions. On the other hand, the existing bronchopneumonia may continue to spread after drainage of the pulmonary abscess. The decision to operate must be based, therefore, on the manifestations in individual cases, but we may state that in general we do not favor operation in the presence of bronchopneumonia of appreciable extent. The same holds true for cases in which two or more pulmonary abscesses exist, for we have operated on a few of these cases and have found that operation did not materially influence the clinical course.

VII. *The Operation.*—Before discussing the technique of the one-stage operation, we repeat our statement concerning the necessity of accurate localization of the abscess. Indeed, we believe that correct surgical approach, which involves entry of the most superficial portion of the abscess through overlying visceroparietal adhesions, is even more important in the case of aerobic than in anaerobic (putrid) abscess. Accordingly, operation should not be performed unless careful roentgenologic study and accurate localization have been carried out. The technique of operation should be, in our opinion, essentially the same as that which we have described for putrid pulmonary abscess.[4] Details therefore will be omitted and only a number of general principles stated. Local anesthesia usually is employed. The incision is placed directly over the abscess, regardless of its location, for there should be no routine incision or favorite site of drainage. Excision of a limited section (two to three inches) of a single rib usually suffices. Edematous visceroparietal pleural adhesions are exposed. They are incised over a small area down to the inflamed lung and are carefully inspected in order to ascertain that the free pleural cavity is completely sealed off within the field. A dry operative field is essential. If the operative exposure is placed at the correct site, aspiration through a thin shell of lung will immediately reveal air, or pus, or both. A specially designed, cutting grooved-director and scissors[5] then are employed to split open the shell of infiltrated lung which comprises the roof of the abscess cavity. In view of the type of infection which exists, as complete an unroofing as in the case of putrid abscess is not necessary. Malleable retractors are inserted, the cavity is sucked dry, and recesses are sought with the aid of direct illumination. If a narrow communication exists between an additional locule and the parent cavity, it is widened by careful sharp dissection. At the termination of operation, the abscess cavity, and any recesses which may exist, are packed snugly to their limits.

The operative treatment of pyopneumothorax, secondary to perforated pulmonary abscess, consists primarily in adequate drainage of

the pleural collection or collections of pus. Special consideration should be given to, and emphasis placed upon, the question of management of the coexisting perforated pulmonary abscess because of the clinical importance of the latter. In the discussion of the surgical pathology of perforated abscess of the lung, attention was called to several features which we have found to be of therapeutic significance as well as of general interest. These have a direct bearing upon the technique of operation. If a pulmonary abscess of substantial proportions characterized by the free expectoration of purulent sputum perforates into the pleural cavity, such perforation may be regarded provisionally as having resulted in adequate drainage if expectoration ceases. If expectoration continues, however, inadequate drainage can be assumed. There can be no difference of opinion up to this point. At the present time, the generally-accepted plan in cases of perforated pulmonary abscess appears to be to limit the operative procedure strictly to drainage of the pleural infection, regardless of the status of the pulmonary abscess. This seems to be based upon the assumption that a pulmonary abscess will heal if perforation into the pleura has occurred. We have observed a number of cases which illustrate the fallacy of this view (see Case 12). It is our opinion that an inadequately draining, perforated pulmonary abscess requires attention at the time of operation on the pyopneumothorax if the patient's condition will permit any procedure beyond emergency pleural drainage. If the latter constitutes the sole measure that can be carried out safely, drainage of the pulmonary abscess should be performed as soon as possible thereafter. The point we wish to stress is that an inadequately draining pulmonary abscess may present a trying as well as an unsatisfactory problem, if its operative treatment is too long deferred. (Cases will be cited to illustrate the contrast between the results of early and late drainage.) The technique of immediate drainage of a perforated pulmonary abscess obviously is simpler than of an unperforated abscess. The sole difficulty may consist in locating the site of perforation. As indicated in our discussion of the pathology of perforated pulmonary abscess, the site of perforation at times may be seen readily, or may have to be sought assiduously. In occasional instances two or more perforations may exist. After the perforation is found, the procedure on the pulmonary abscess is limited to simple crucial incision through its roof, at the site of perforation, followed by packing of the pulmonary cavity.

VIII. *Postoperative Course and Treatment.*—The postoperative course of cases of unperforated abscess of the lung usually is smooth if the pulmonary abscess is the sole lesion; and stormy if there is associated necrosuppurative bronchopneumonia. The existence of the latter is indicated by continuing cough, purulent expectoration, and

fever. Distant areas of bronchopneumonia also may be the source of these manifestations. The point to be stressed is that, in contrast to cases of putrid abscess of the lung, untoward postoperative manifestations often may be referable to the underlying disease (necrosuppurative bronchopneumonia) rather than to complications in the operative field. The management of the wound usually is simple. Packings are removed when they are loose (ordinarily in a week's time) and are replaced in order to prevent premature closure of the cavity. At each succeeding dressing the cavity is packed lightly, until it has become obliterated completely. The superficial wound then is permitted to heal.

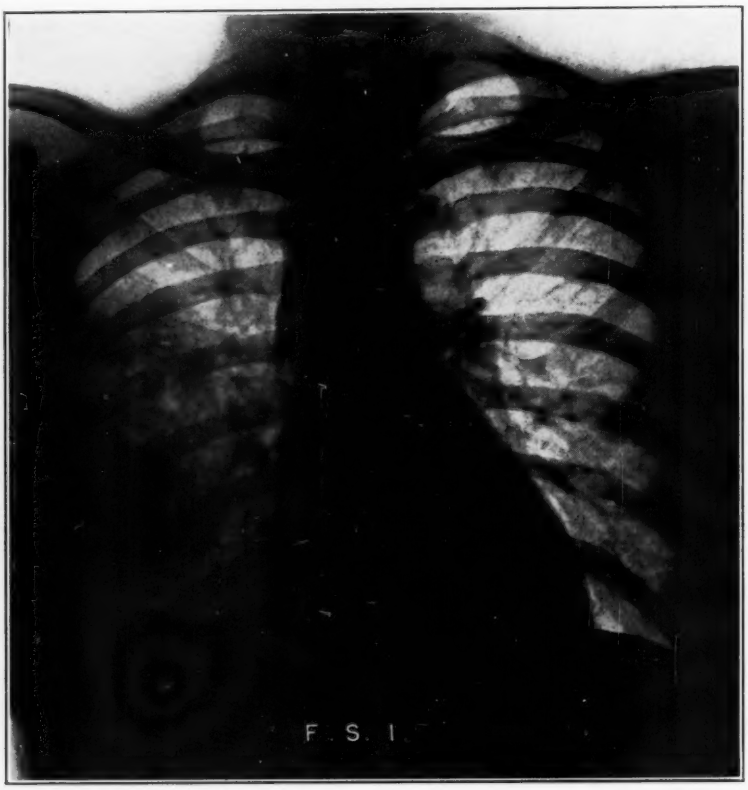

Fig. 2.—F. S. Necrosuppurative bronchopneumonia of right lower lobe. Note multiple areas of rarefaction. This type of lesion characteristically precedes aerobic pulmonary abscess.

A "lattice lung" may be the sequel when numerous bronchial fistulae are present in a large smooth-lined cavity and is apt to remain chronic. As pointed out elsewhere,[6] free grafts of fat have been transplanted successfully for the obliteration of pulmonary cavities even in the early postoperative period. Regardless of the method that is chosen, every effort at closure of the pulmonary cavity should be made as soon as the infection has cleared up.

IX. *Results of Treatment.*—The results of operation for pulmonary abscess can be indicated most readily by dividing the cases into groups

in accordance with special features of the pathologic process. Although many variations of the latter have been indicated already, for purposes of presentation the cases will be divided into only a few general groups. Illustrative cases, selected from each group, will demonstrate the problems which have been encountered and the results that have been obtained. Interest, from a surgical viewpoint, attaches chiefly to the results in the first group, and most of the operative cases in this group therefore will be reported. Only a few illustrative cases from each of the other groups will be reported.

Group I. Acute Pulmonary Abscess.—All cases were operated upon within six weeks of the onset of pulmonary manifestations, the majority being operated upon in the second and third weeks. All are well, the follow-up period ranging from one to several years.

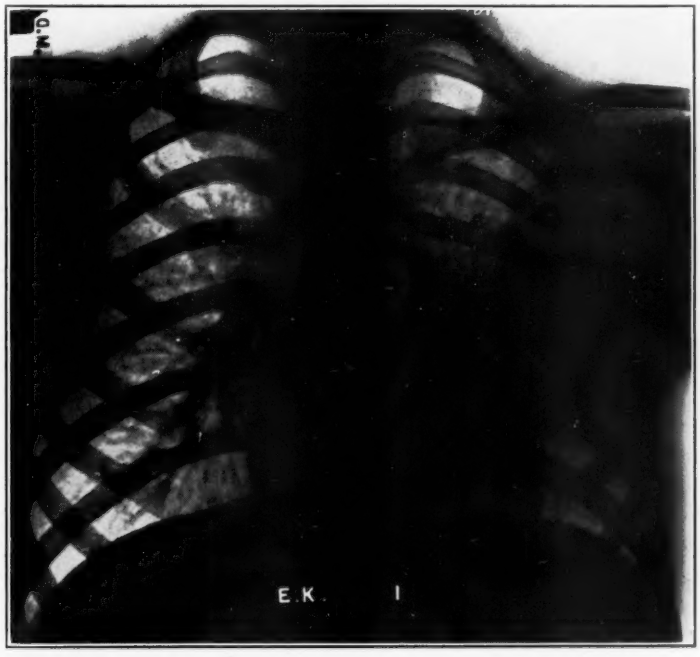

Fig. 3.—E. K. Necrosuppurative bronchopneumonia in left upper lobe. Note density of pulmonary infiltration.

CASE 1.—M. R., female (Hospital No. 386,380), 20 years old, admitted November, 1935, was ill for eleven days prior to admission with chills, fever, cough, expectoration of pus, and severe toxemia. The roentgenogram disclosed a large cavity, with fluid level, in the left upper lobe. The patient became worse during ten days of hospital observation, with increasing amounts of sputum and roentgenologic evidence of increase in the size of the lesion. Bronchoscopy was performed because of doubt between the diagnosis of shut-off putrid abscess and that of tuberculous cavitation. It revealed nonfetid pus issuing from the left upper lobe bronchus. The diagnosis of aerobic pulmonary abscess having been made, operation was decided upon because the clinical course obviously was unsatisfactory. At operation, an abscess in the

upper lobe, situated adjacent to the interlobar fissure, was unroofed and packed. Fever, as well as some purulent expectoration, continued for three weeks after operation due to an area of necrosuppurative bronchopneumonia in the opposite lung. This fact was established by bronchoscopy and by x-ray. The wound remained in good condition. Postoperative bronchoscopy, performed in order to determine if retention of pus in the operative field was the cause of continuing fever, was essentially negative. Convalescence was slow. A bronchial fistula was present in the depths of the wound for several weeks after operation. It was permitted to close after x-ray films of the chest revealed that pulmonary infiltration was subsiding. The wound has remained healed; the patient is symptom-free; x-ray of the chest is negative.

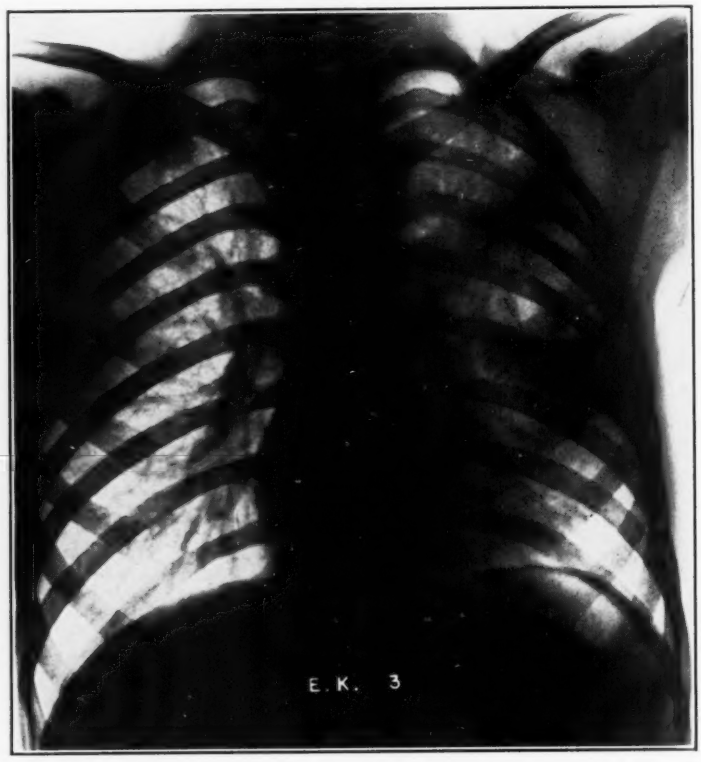

Fig. 4.—E. K. Same case as shown in Fig. 3, several days later. Typical abscess replacing area of pulmonary infiltration. There should be noted the long fluid level suggesting pyopneumothorax as well as the limited surrounding infiltration.

Case 2.—S. W., male (Hospital No. 394,118), 12 years old, admitted April, 1936, was ill with "pneumonia" for four weeks before admission. There were fever, expectoration of moderate amounts of thick pus, and, in the roentgenogram, a dense, homogeneous shadow occupying the region of the right lower lobe. A few days before admission, a large amount of pus suddenly was expectorated. The x-ray film then revealed a giant cavity with fluid level. There was a difference of opinion as to whether the lesion was confined to the lung or had perforated into pleura. Operation was performed shortly after admission because the septic course had continued despite partial evacuation of the abscess by way of the bronchial tree. At operation, a thin shell of avascular lung was traversed through well-defined pleural adhesions.

An enormous pulmonary cavity was encountered. It occupied much, if not most, of the lower lobe, was smooth-lined, partly empty, and presented many large and small bronchial orifices situated on and between fleshy ridges. Communicating with this main cavity was a small abscess situated within the lobe just above the diaphragm. The bridge between the two was enlarged by sharp dissection, pus in the smaller abscess was removed by suction, and both cavities were packed.

The patient was afebrile within three days of operation and remained so. There was prompt cessation of cough and expectoration, and rapid improvement in general condition. The pulmonary cavity quickly became clean. It soon assumed the appearance characteristic of lattice lung. Because of a distinct tendency towards healing, the skin was permitted to close. The patient was symptom-free, but x-ray films demonstrated persistence of the cavity. Accordingly, the wound was reopened

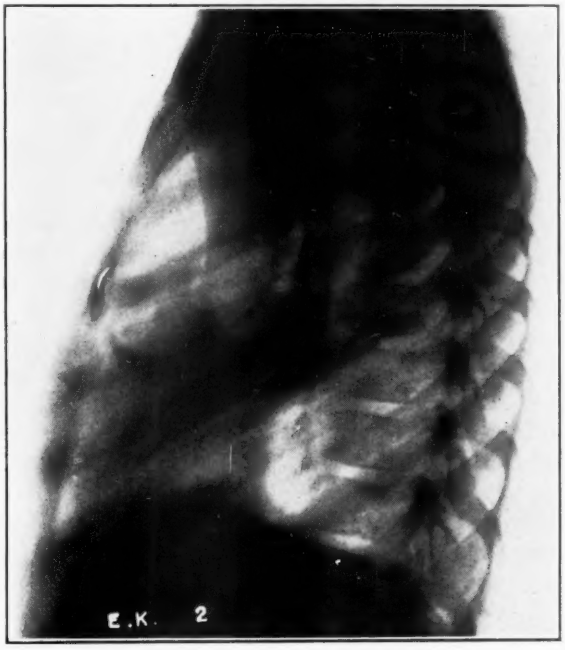

Fig. 5.—E. K. Lateral view, filmed at same time as Fig. 4. The abscess is situated in the axillary portion of upper lobe close to the fissure. An analysis of Figs. 4 and 5 offers a precise localization of the lesion.

about two months after the original operation and a free graft of fat was introduced.[6] The patient has remained well and the cavity is closed.

CASE 3.—A. S., male (Hospital No. 405,565), 13 years old, admitted March, 1937, with a history of eleven days' severe illness. In the beginning, meningitis was suspected. Pneumonia of the right lung was diagnosed on the third day; cough and expectoration of pus began soon thereafter. Expectoration of pus in increasing amounts led to the suspicion of a pulmonary abscess. The diagnosis of pyopneumothorax was made on the preadmission x-ray films. Fever was high and septic, and the patient was critically ill throughout the eleven days.

Films taken on admission to the hospital indicated a large abscess, with fluid level, situated in the posterior part of the right upper lobe and confined to the lung. Operation was performed on the day of admission. Adherent, inflamed lung was exposed by the excision of a short section of one rib. Aspiration through a thin

layer of lung revealed pus and air. The abscess was unroofed by a rather liberal excision of the overlying avascular shell of lung (no more than one-fourth of an inch in thickness). The pus was evacuated by suction, the cavity found to be monolocular with several moderate-sized bronchial fistulas. The cavity was packed with gauze.

The postoperative course was entirely smooth at first, with prompt disappearance of cough and expectoration and subsidence of fever. Later, however, there was recrudescence of fever without cough. The roentgen film revealed pulmonary infiltration adjacent to the abscess cavity and distinct widening of the mediastinum. One week later, a film disclosed subsidence of this newly developed infection, and subsequent films demonstrated the complete disappearance of pulmonary infiltration and of evidences of mediastinitis. The wound always was in satisfactory condition, and quickly contracted about the tube leading to the region of the bronchial fistula. The tube was removed when the x-ray films indicated the pulmonary condition to be satisfactory, and the wound then healed promptly. The patient has remained symptom-free.

CASE 4.—E. K., male (Hospital No. 390,519), 13 years old, was ill for nine days before admission in March, 1936. At the onset the diagnosis of left upper lobe pneumonia was made and was confirmed by a film disclosing a dense shadow occupying about half of the upper lobe. Pain in the chest was a prominent feature. Expectoration of pus began four days after the onset, and rather large amounts were brought up each day. The level of fever was lower after the first week. X-ray films taken two days before admission revealed a large cavity occupying much of the site of the pre-existing pulmonary infiltration. On hospital admission, films showed an increase in the size of the cavity and suggested that perforation into the interlobar fissure had occurred. The assumption of perforation was the indication for prompt operation. The operation, carried out through a surgical approach in the left axilla, consisted in the removal of a short section of one rib, disclosing agglutinating visceroparietal pleural adhesions, and the entry of a large cavity in the upper lobe through these adhesions. The overlying thin shell of lung was excised and the interior of the cavity inspected. Several small bronchial fistulas were noted. Although actual perforation into the interlobar fissure had not occurred, the adjacent lower lobe could be seen through the floor of the abscess cavity and perforation undoubtedly was imminent.

Clinically, the postoperative course was smooth and entirely uneventful. There was prompt cessation of cough and expectoration. However, the postoperative film showed pneumonic spread about the abscess cavity, which soon subsided. Early closure of the wound was permitted because the postoperative bronchial fistula was small.

The patient was well until a year and a half after operation (December, 1937), at which time he contracted a right upper lobe pneumonia. A roentgen film revealed areas of rarefaction in the midst of pneumonic infiltration and the diagnosis of a necrosuppurative bronchopneumonia was made. Spontaneous subsidence occurred. It was of interest to note that the left lung remained free from invasion. The patient has remained well since that time.

CASE 5.—B. D., male (Hospital No. 379,070), 18 months old, entered the hospital in April, 1935, with a history of upper respiratory infection, cough, fever, and convulsions of five days' duration. Roentgenography revealed a pleural effusion occupying the axillary portion of the right chest from base to apex. Aspiration yielded slightly turbid, yellowish fluid. Over a period of approximately two weeks, there was

little change to be seen in successive roentgenograms. The pleural fluid, which was aspirated several times, did not increase in amount. At the end of the two-week period, however, a film disclosed not only an increase in the pleural effusion but also two cavities, containing fluid levels, within the middle lobe. There was very little pulmonary infiltration about the cavities. At operation, a small amount of encapsulated turbid fluid was evacuated from the pleura and the abscesses in the middle lobe were evacuated, unroofed, and packed. Convalescence was uneventful. The patient has remained well.

The following case, which first came under our observation many months after operation for acute pumonary abscess, is presented as an illustration of an unsatisfactory result after inadequate drainage.

CASE 6.—W. M., female (Hospital No. 417,402), 23 years old, was ill for five weeks with cough, purulent expectoration, and fever, at the time of her first operation elsewhere in June, 1937. In the preoperative film there was seen a multilocular lesion in the right lower lobe, with limited surrounding infiltration. This intrapulmonary collection of pus was drained through a small incision. Purulent expectoration continued, a second limited rib excision was performed at a somewhat higher level, and the pulmonary cavity was again entered and drained by a tube. Cough and purulent expectoration continued unchanged, and drainage from the pulmonary cavity continued. This was the status on admission to Mount Sinai Hospital where a chronic, multilocular abscess was liberally unroofed. Expectoration of pus subsided soon thereafter. The pulmonary lesion, however, was a chronic lattice lung which demonstrated no tendency to heal. It was too large and discharged too profusely to warrant an attempt at plastic closure. Accordingly, lobectomy was performed. After a long and trying postoperative period, the patient appeared convalescent for a time. Death occurred quite suddenly several months after operation apparently due to heart failure since no other cause was found at autopsy.

Group II. Subacute and Chronic Abscess.—These lesions are rare, because the antecedent acute abscess usually either subsides spontaneously, is fatal, or leads to pleural complications for which some operative procedure is performed. The following are the only two unperforated subacute or chronic aerobic abscesses which have come under our observation:

CASE 7.—B. L., male (Hospital No. 380,596), 42 years old, was first admitted in 1932 with a six years' history of recurring episodes of pain in the chest, cough, purulent expectoration, and transient fever. The original illness was said to have been a bronchopneumonia, and each of the episodes in succeeding years involved the same region of the lung. On admission, the roentgenogram revealed an abscess cavity in the axillary segment of the right upper lobe with little surrounding pulmonary infiltration. At the primary operation, a very stiff-walled pulmonary abscess about one and one-half inches in diameter was unroofed. There were no recurrences of the episodes of cough, fever, and chest pain, but profuse discharge from the cavity continued during the succeeding years. Secondary operations, for the purpose of establishing better drainage, were performed on two occasions, but did not result in improvement. Finally, resection of the upper lobe was performed three years after the original operation, for the purpose of eradicating the disease. This was followed by death from acute fibrinous (obstructive) bronchitis.

CASE 8.—L. H., male (Hospital No. 410,785), 52 years old, was admitted to the hospital in June, 1937, with a history of fever, cough, and profuse purulent expectoration of seven weeks' duration. Three days before admission, a large abscess developed in the soft parts over the right chest anteriorly. This was drained on admission. Roentgenography revealed an area of infiltration in the apex of the left lower lobe, which suggested the diagnosis of bronchial neoplasm with secondary pulmonary suppuration. Bronchoscopy, however, failed to reveal the presence of a bronchial neoplasm. There was no appreciable change in the successive roentgen films taken during a period of three weeks. At the end of that time, however, a

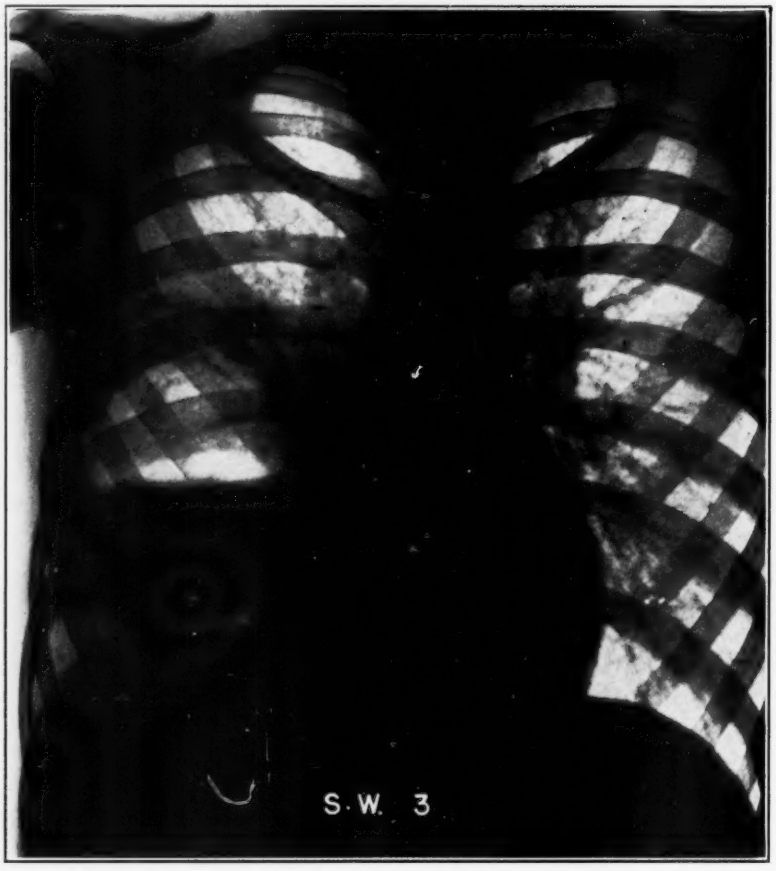

Fig. 6.—S. W. Enormous, typical pulmonary abscess confined to the right lower lobe. Limited surrounding pulmonary infiltration. Long fluid level strongly suggestive of pyopneumothorax.

large area of cavitation containing air and fluid appeared in the midst of the infiltrated area. At operation, a large unilocular nonputrid pulmonary abscess was evacuated, unroofed, and packed. Although the temperature became lower and cough and expectoration diminished, improvement was only temporary and subsequent films disclosed further extension of pulmonary infiltration. At the end of nine and one-half weeks, cavitation became visible within the infiltrated area; and at operation two large contiguous abscess cavities were evacuated, unroofed, and packed. Following this procedure, uneventful convalescence ensued. The patient has remained well.

Group III. Necrosuppurative Bronchopneumonia Complicated by Pulmonary Abscess.—Cases in this group are apt to go on to fatal termination, being uninfluenced by drainage of one or more pulmonary collections of pus. The latter usually are not definitely encapsulated. Empyema may be an added complication, and its drainage also is not likely to influence the clinical course favorably. Of this type of case, one example of an unperforated and one of a perforated lesion will be given.

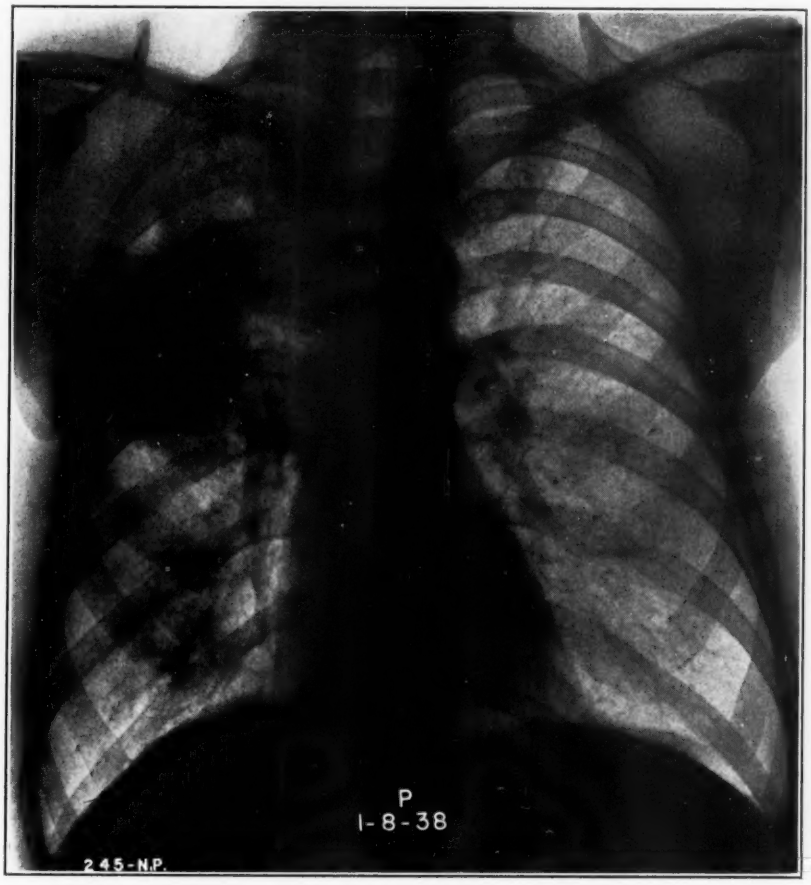

Fig. 7.—P. Large pulmonary abscess with localized pyopneumothorax. The overlapping fluid levels of each of these lesions is seen in the axillary portion of the dense homogeneous shadow.

CASE 9.—S. C., male (Hospital No. 417,451), 54 years old, was ill for seven weeks when admitted. The course throughout this period was characterized by irregular fever ranging to 104°, cough, profuse purulent expectoration, and evidence of toxemia. Roentgen films revealed large areas of infiltration in the right lower lobe, in two separate regions. Operation was performed on the diagnosis of perforated pulmonary abscesses with encapsulated empyemas. The lesions were approached through two separate incisions. At each site multilocular collections of pus were evacuated. They lay superficially within the substance of the lung and were entered

through well-defined pleural adhesions. There was no empyema. In each instance, the multiple collections of pus were not encapsulated and the adjacent lung obviously was the seat of an intense pneumonitis.

There was transient improvement for about two weeks, and then a return to the preoperative state. Death occurred about five weeks after operation due to spread of the infection throughout much of the right lung and part of the left.

CASE 10.—C. P., male (Hospital No. 418,725), 58 years old, was ill for two months before admission. The clinical course was very similar to that in the preceding case. An early film revealed dense infiltration in the right upper lobe; later films demonstrated areas of rarefaction within that shadow. He was operated upon shortly after admission on the diagnosis of perforated pulmonary abscess. An extensive multilocular suppurative process, involving much of the upper lobe, was encountered. Purulent tracts were found extending in various directions in the midst of intensely inflamed lung. There were two perforations which led to substantial collections of pus in the pleura.

Expectoration of pus diminished temporarily after operation and then recurred. Subsidence of fever and improvement in the general condition also were transient. Death occurred three weeks after operation from continuing pulmonary suppuration and cerebral infection. At autopsy, collections of pus adjacent and distant to those which had been drained, were found in the midst of bronchopneumonic areas. Multiple abscesses of the brain also were present.

Group IV. Perforated Solitary Pulmonary Abscess.—A variety of lesions fall into this group, but do not lend themselves to subgrouping. Two examples are chosen, not so much to illustrate the pathologic features as to point to the results of logically conceived treatment in one case and incorrect treatment in the other.

CASE 11.—G. G., male, 40 years old, was ill for two weeks when admitted to the Hospital for Joint Diseases. At the onset there were severe pain in the right chest, fever, and cough. Purulent expectoration began soon thereafter and became profuse. An early film disclosed a circular shadow in the right lower lobe, which was obscured in a later film by the homogenous shadow of a pleural effusion. Aspiration of the chest revealed pus. At operation a large empyema, situated posterolaterally, was entered and evacuated. The perforation of a pulmonary abscess was visualized. It was enlarged, and an abscess cavity of substantial proportions was unroofed. Expectoration of pus ceased promptly after operation and did not recur. Subsequently an anterior, parapericardial empyema, derived from an interstitial perforated pulmonary abscess, was drained. These two empyemas were separate and distinct from each other. Finally an encapsulated pleural infection in the right axillary region was opened and was found to be derived from an adjacent small perforated interstitial abscess. It was unrelated to either of the two previously mentioned perforated abscesses. Complete recovery followed the third operation. Of the three perforated abscesses in this case, only the first, characterized by the expectoration of pus, was of the segmental type which is the subject of this paper.

CASE 12.—E. N., female (Hospital No. 375,996), 30 years old, was operated upon in March, 1933, for a pneumococcus pyopneumothorax. At first, closed drainage was performed. After the acute phase had subsided, an open thoracotomy for the residual empyema was performed. Cough, purulent expectoration, and fever persisted for several weeks after operation, and then all manifestations gradually subsided. The wound healed slowly, a bronchial fistula being present for two months.

The patient appeared to be entirely well in July, 1933, at which time she was operated upon for cholecystitis and cholelithiasis. There were no symptoms referable to the chest during her convalescence.

In October, 1933, occasional cough, purulent expectoration, and small hemoptyses began. The patient entered the hospital one year later (November, 1934), shortly after a large hemoptysis. A bronchogram revealed a cavity in the right upper lobe. Operation was performed, and a chronic abscess of the lung unroofed. A bronchial fistula, perforation into the pleura, and chronic encapsulated empyema were found. The pulmonary abscess was the original one, which had not been sought and cared for at the operations performed for the pleural infection in 1933. The operative procedure was difficult and a revision, for better drainage of the pulmonary cavity, was required in 1935. The patient has been well since that time, and a bronchogram demonstrates a normal bronchial tree.

Although not noted in all cases, there are three distinctive features of pulmonary abscess in children: (1) unusually early and extensive destruction and excavation of the lung, (2) early invasion of the pleura, (3) absence of expectoration of pus, even in older children. Examples of perforated solitary pulmonary abscess will be given to illustrate (1) the problem of management of a perforated pulmonary lesion with limited empyema, (2) the correct and incorrect treatment of perforated pulmonary abscess with total or subtotal pyopneumothorax.

CASE 13.—I. H., male (Hospital No. 364,367), 11 months old, was admitted in April, 1934, with a history of pneumonia of the right upper lobe of one week's duration. He was under hospital observation for eleven days, during which time there were fever, cough, and roentgen evidence of a progressively destructive lesion of the right upper lobe. By the time the patient came to operation, a very extensive lesion involving most of the upper lobe, with multiple fluid levels, was seen in the x-ray film. The operative procedure consisted of the drainage of an encapsulated pyopneumothorax and the enlargement of the pulmonary opening. The enormous multilocular pulmonary cavity, with its numerous bronchial fistulas, was converted essentially into a single cavity by the division of septa, and was packed.

There was prompt subsidence of clinical evidence of suppuration, and the cavity was clean two weeks after operation. At that time, autotransplantation of fat into the cavity was performed in the hope that the difficult problem of lattice lung would be obviated. The wound healed over the graft, the child has remained symptom free, and x-ray check-ups of the chest in the past four years have disclosed no abnormalities.

CASE 14.—R. B., female (Hospital No. 415,912), 11 months old, was admitted in October, 1937, and was under observation for two weeks with the diagnosis of pneumonia followed by empyema. Aspiration of the chest revealed pus. The only reason for suspecting the existence of a pulmonary abscess was the presence of areas of rarefaction within the shadow of pneumonic infiltration. As proved by operation, the abscess would have been overlooked if, on the basis of the roentgen findings, it had not been especially sought. The bilocular empyema, situated in the axilla, was first laid open. Its anterior limits were then explored because the suspected pulmonary abscess was localized in the anterior axilla. A narrow purulent tract was followed and found to enter an empyema which led to the site of perforation of a pulmonary abscess of substantial proportions. After the removal of a

section of an additional rib, the pulmonary abscess as well as the empyema could be adequately unroofed. Both cavities were then packed. Convalescence was uneventful, and there was early obliteration and healing of both cavities. The perforated pulmonary abscess was undoubtedly the source of the empyema in this case. However, the communication between them consisted of a narrow tract which had to be sought. Its discovery and the resultant surgical management of the pulmonary abscess comprised a simple solution of a problem which might have proven difficult if deferred to a subacute or chronic phase. Furthermore, the complications of a persistent, untreated pulmonary abscess were eliminated by early adequate drainage.

CASE 15.—R. M., male (Hospital No. 386,733), 19 months old, was admitted in November, 1935. The first operation consisted of closed drainage of a large, tense

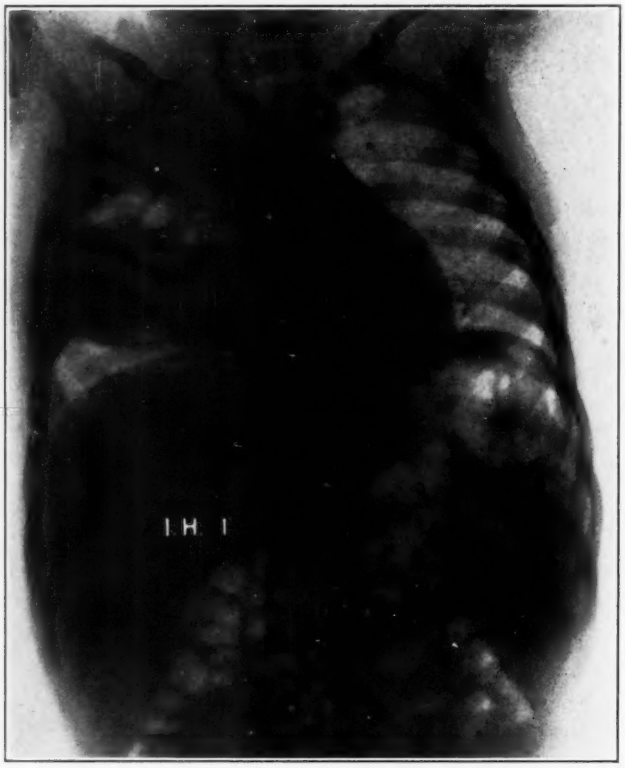

Fig. 8.—I. H. Extensive pyopneumothorax encapsulated over upper lobe. Note multiple fluid levels lying partially within the lung and partially within pleura.

pyopneumothorax at a time when the patient was in a critical condition. There was prompt improvement. Enormous quantities of air escaped for days through the underwater system, thus offering evidence of a perforated pulmonary abscess of substantial proportions. With the subsidence of the hyperacute clinical manifestations, consideration was given to the fact that a large perforated pulmonary lesion existed and was associated with a total or subtotal pneumothorax. Accordingly, a definitive operation was decided upon five weeks after the primary one, with the object of closing the large pulmonary defect. The lesion, exposed by the excision of sections of two ribs, consisted of a typical lattice lung, presenting a number of major bronchial fistulas. The lung adjacent to the blown out abscess was adherent. It was freed in part, and was sutured over a fat graft. There was some leakage of

air from the operative site, with resultant postoperative pneumothorax. The latter cleared up slowly. The lung has remained expanded and free from pulmonary infiltration.

CASE 16.—R. B., male (Hospital No. 399,494), first came under observation in 1932 at the age of 5 years, and the nature of the problem best will be appreciated when attention is called to the fact that the child was not well until the end of 1937, after a long series of hospital admissions and operations. When admitted in 1932, there was a history of pneumonia in 1930 followed by empyema and coinci-

Fig. 9.—I. H. Lateral view. The multiple pyopneumothoraces are confined to the anterior and axillary portion of the thorax. The necessity of lateral films for localization even of large encapsulated lesions is evident.

dental expectoration of purulent material. The pleural infection was treated by repeated aspiration and injection of air. This resulted in complete collapse of the lung and persistent pneumothorax. Since that time there were recurrent attacks of fever and cough. Shortly after admission to Mount Sinai Hospital, exploratory thoracotomy was performed. The lung was found collapsed and fixed against the mediastinum; there was a large pulmonary excavation in the lower lobe with the characteristic features of lattice lung, and purulent exudate was present over the adjacent pleura. Sections of several ribs were excised for purposes of direct drainage of the pulmonary lesion. A number of thoracoplastic operations were performed upon subse-

quent admissions, in order to obliterate the large pleural dead space. They succeeded only partially, and the lattice lung persisted. Finally, in order to eradicate the lesion, removal of the lower lobe was performed. A bronchial fistula persisted, which was closed by means of a fat graft. The patient has remained well since that time.

Group V. Perforated Multiple Pulmonary Abscesses.—As already indicated, these cases, occurring most often in children, are apt to be fulminating and fatal in their course, and usually do not present

Fig. 10.—F. Widely separated perforated pulmonary lesions with multiple encapsulated pyopneumothoraces. The latter are more evident in the lateral than in the posteroanterior film.

problems for surgical management other than emergency measures for temporary palliation. The following are unusual instances in which operative treatment was indicated.

CASE 17.—L. B., female (Hospital No. 410,035), 23 years old, was admitted in April, 1936. The illness began six weeks before, with fever, chill, and chest pain, and the expectoration of bloody sputum. Subsequently the sputum became purulent. The diagnosis of bronchopneumonia followed by "pleurisy" was made. She was in the hospital for ten days with high fever, dyspnea, cyanosis, and rather purulent expectoration. Only after several exploratory aspirations was purulent fluid encountered in the pleura. At operation, a most extensive multilocular empyema was revealed. Separate collections of pus reached from the posterior aspect of the

apical portion of the lung down to the diaphragm. In addition, there was an infrapulmonary empyema which could be evacuated only after detachment of the adherent lower lobe from the diaphragm. There were several underlying perforated pulmonary abscesses, which were split open by crucial incisions. Pulmonary and pleural abscess cavities were packed. Fever and some purulent expectoration continued for six weeks after operation, apparently due to areas of necrosuppurative bronchopneumonia. The extensive wound progressed satisfactorily. A sinus persisted for many months, but finally healed. The patient has been entirely well and symptom-free. The bronchogram was normal except for the crowding together of several of the bronchi of the lower lobe.

CASE 18.—M. H., female (Hospital No. 348,206), 38 years old, was admitted in January, 1933, with a four weeks' history of bilateral bronchopneumonia. The lesions appeared almost simultaneously in both lungs and were characterized by chest pain, fever, cough, and purulent expectoration. The latter was progressive, but fever tended to subside. Shortly before admission, collapse phenomena supervened. An x-ray film, taken directly after admission, revealed multiple bilateral pulmonary abscesses with perforation of one of them and consequent pyopneumothorax in the right axilla, and an empyema on the left side. The first operation was performed on the right side. A bilocular pyopneumothorax, derived from a perforated lung abscess, was unroofed. One week later, the left-sided bilocular empyema was drained. This, too, was found to be derived from a perforated pulmonary abscess. The postoperative course was satisfactory and was characterized by spontaneous healing of all of the unperforated pulmonary cavities. The patient has remained well, and a recent roentgenogram (1938) is essentially negative.

SUMMARY AND CONCLUSIONS

Segmental aerobic abscess of the lung is a complication of necrosuppurative bronchopneumonia. The latter term was coined because the coexistence of suppuration and necrosis characterizes the disease. The pathology, and the clinical and roentgenographic features of necrosuppurative bronchopneumonia are described.

Acute aerobic abscess of the lung is no longer a rare lesion because the incidence of necrosuppurative bronchopneumonia has greatly increased. The stage of transition between necrosuppurative bronchopneumonia and aerobic pulmonary abscess is not recognizable clinically.

Aerobic pulmonary abscess is seen in two important forms: interstitial and segmental. The latter alone is the subject of the present communication. Of the two forms in which it occurs (the multiple and the solitary), the solitary abscess is usually the only variety which is of surgical significance.

Three classes of solitary abscess are described: (1) pulmonary abscess as an incidental part of an extensive necrosuppurative bronchopneumonia; (2) pulmonary abscess as the predominating lesion within an extensive area of necrosuppurative bronchopneumonia; (3) typical pulmonary abscess surrounded by a more or less sharply limited and narrow zone of infiltrated lung.

There are pathologic features which are common to all three varieties: substantial size, monolocularity, superficial situation within a pulmo-

nary lobe, overlying agglutinating pleural adhesions, more or less extensive destruction of bronchi.

The clinical manifestations of segmental pulmonary abscess are essentially indistinguishable from those of the antecedent necrosuppurative bronchopneumonia. The differentiation must be based on roentgenographic findings. Pulmonary abscess must be differentiated from other pulmonary diseases, also, especially tuberculosis and neoplasm.

The indications for operative treatment, which have as yet not been completely formulated, are discussed in detail. Correct operative approach is based upon accurate preoperative localization of the lesion. The operative method which is advocated is a single stage procedure. Details of the operation for perforated and unperforated lesions are given. Postoperative treatment is described.

The results of operation in the three varieties of aerobic abscess, as well as in perforated abscess, are indicated in the case reports. Operation for pulmonary abscess in Classes 1 and 2 has been unsatisfactory. Operation for abscess in Class 3 has been followed by cure in every instance.

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INCIDENCE OF AIR-BORNE BACTERIA IN THE MAJOR SURGERY OF THE MULTNOMAH COUNTY HOSPITAL

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THE PRESENCE of air-borne bacteria is at present considered inescapable in any peopled room. Nevertheless, a surgery cannot be viewed as any ordinary room. The incidence of bacteria in the air of such a room is a matter of professional concern to the surgeons and of vital concern to the patient. Surgeons during the Listerian era of antiseptic surgery took elaborate precautions to minimize this source of sepsis. Although no phenol sprays are in use today, just as elaborate precautions are observed in decreasing the sepsis from other sources, the surgeons rightly considering air-borne contamination of relatively minor moment. Aseptic surgery has derived its lease on efficacy through rigid adherence to bacteriologic principles. With the almost ritualistic refinements in surgical technique eliminating previous major sources of sepsis, the question of air-borne contaminants is promoted to a status of major importance. The contemporary surgical literature gives token of awakened interest in this subject.[1-14] For this reason an investigation of the incidence of air-borne bacteria in the major surgery of the Multnomah County Hospital was considered to be of interest.

EXPERIMENTAL PROCEDURES

Fresh human oxalated blood was used in the preparation of blood agar plates. With each set of ten plates so prepared, two control plates were made and incubated concurrently. The plates were then retained in the refrigerator of the Multnomah County Hospital surgery until an opportunity for exposure presented itself. The plates were placed in designated spots and marked according to their disposition, in accordance with a map of the major surgery (Fig. 1). Exposure was for a constant period of 30 minutes. At the expiration of this interval, the plates were covered, collected, and incubated at 37° C. A count of the colonies was made at 24, 48, 72, and 96 hours. After three sets of plates had been so incubated, however, it was found that incubation longer than 48 hours resulted in a spreading overgrowth of the colonies in a fashion making an accurate count impossible. As a result of this, only the figures obtained at 48 hours were considered significant. At no time during the period of incubation were the covers of the Petri plates lifted. When the designated period of incubation had been passed, the

Received for publication, June 1, 1938.

covers of the plates were lifted and identification of the colonies attempted. Exact determination of the separate colonies was considered beyond the scope of this paper. From a naked-eye examination of the colonies, a fairly accurate identification of the streptococcus and staphylococcus colonies could be made. A sufficient number of small hemolytic staphylococcus colonies were checked microscopically to insure reasonably accurate differentiation from the streptococcus colonies. No attempt was made beyond this. If a colony did not present the characteristics of the organisms named, it was classed with the unidentified group. This latter would include, undoubtedly, such organisms as *Proteus vulgaris, Eschirichia coli, Neisseria catarrhalis, Bacillus subtilis,* and other common bacteria.

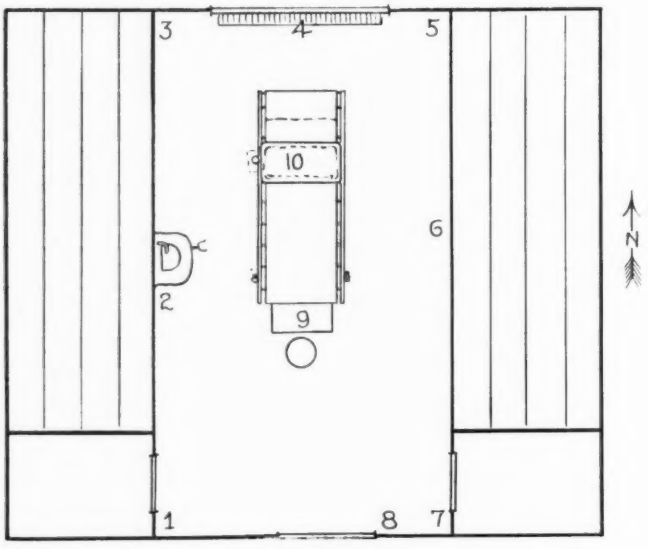

Fig. 1.

RESULTS

The results obtained from this investigation are summarized in the accompanying tables and charts. The contributing factors to an increase in the number of air-borne contaminants is well illustrated in Table I and shown graphically in Fig. 2. Under basal conditions at night with the surgery unentered, there is a relatively low incidence of bacteria. In the day, even though the surgery was unentered during the actual exposure time, the air had been sufficiently agitated to produce a slight rise in the count. When the plates were exposed with the doors left open and people entering, there was a significant rise in the bacterial count. The source of this increase may be explained by: (a) exogenous bacteria brought in from outside the surgery in the wake of the entrants, (b) endogenous bacteria wafted from their previous resting places in the

breeze created by the movements of those entering, or (c) contaminants from the nasopharynx of the unmasked entrants. This count is by no means as significant as the counts found on plates exposed during operative procedures. A sharp rise in the number of bacteria is noted immediately upon putting the surgery to its proper use. This mounts steeply when one section of ten students is placed in the balconies. Fig. 3

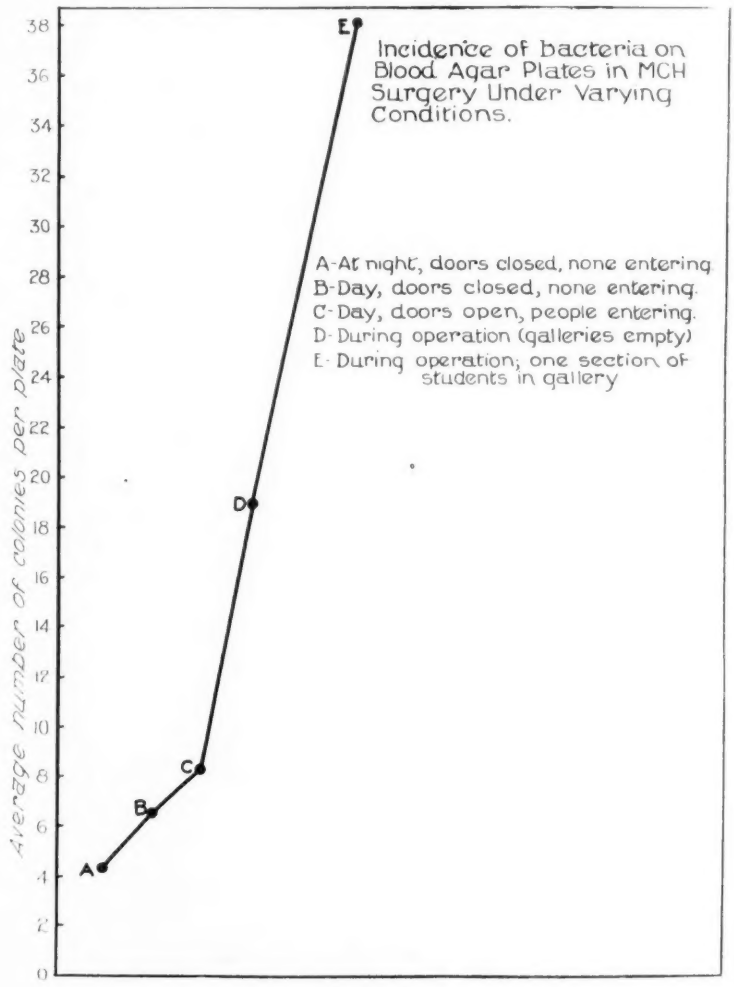

Fig. 2.

shows graphically the increase in the type of organism. It is to be noted that no hemolytic streptococci are found until operative procedures are undertaken. At this time both the streptococci and the *Staphylococcus albus* show a marked increase. Table III shows the estimation of the number of bacteria falling on a sterile operating field in one hour as estimated by various workers. Meleney[2] estimated that a Petri plate covered 7 square inches and that the usual operating field covered an area of

TABLE I

NUMBER OF BACTERIAL COLONIES ON BLOOD AGAR PLATES INCUBATED AT 37° C. FOR 48 HOURS AFTER A 30-MINUTE EXPOSURE IN LOCATIONS DESIGNATED BY NUMBER IN FIG. 1

PLATE NUMBER	CONDITIONS OF EXPOSURE*				
	A	B	C	D	E
1	7	6	10	26	164
2	3	9	12	23	58
3	4	4	6	25	43
4	3	6	7	26	47
5	5	4	30	27	49
6	1	5	3	11	38
7	5	8	8	13	32
8	6	11	12	21	30
9	5	5	8	12	52
10	4	8	12		

*A—At night with doors closed and no one entering surgery. B—During day with doors closed and no one entering surgery. C—During day with doors open and people entering surgery. D—During course of day's operations with balconies empty. E—During operation with one section of students in balconies.

from 4,000 to 7,000 square inches. This included the area occupied by the sterile instrument tray, the actual operating field, and the rinse basins. It is from these figures that all estimations are made.

TABLE II

AVERAGE BACTERIAL COLONY PER PLATE UNDER CONDITIONS OF EXPOSURE AND INCUBATION DESCRIBED IN TABLE I

BACTERIAL TYPE	AVERAGE NUMBER OF COLONIES PER PLATE*				
	A	B	C	D	E
Hemolytic streptococcus	0.0	0.0	0.0	4.2	5.0
Nonhemolytic streptococcus	0.0	0.5	0.8	5.2	5.5
Staphylococcus albus	2.2	3.4	3.7	9.2	16.0
Staphylococcus aureus	1.1	0.1	1.1	1.7	3.5
Fungi	0.1	0.7	0.0	0.25	0.6
Not identified	0.1	0.7	0.3	9.7	18.3
Overgrown plates	0.0	0.0	2.0	5.0	3.0
Total colonies (all)	4.3	6.6	8.3	19.0	37.3

*The identification under conditions B, C, and D was made after the plates had been incubated for 96 hours.

COMMENTS

As previously stated in this work and as emphasized by Gardner,[1] Meleney,[2] and Hart,[4] when the improvement in surgical technique reduces other sources of sepsis to a minimum, then by comparison air-borne

TABLE III

NUMBER OF BACTERIA FALLING ON STERILE OPERATING FIELD IN ONE HOUR AS ESTIMATED BY VARIOUS WORKERS

AUTHORITY	NUMBER OF BACTERIA	COMMENT
Meleney	35,000-60,000	
Davis	19,000-35,000	
Hunt	44,000-78,000	"Old-fashioned" surgery
Our estimate	21,000-38,000	Usual operation galleries empty
Our estimate	42,000-74,000	One section students in galleries

contamination becomes a question of major concern to the surgeon. From this study it does not appear that air-borne contamination is at an irreducible minimum in the major surgery of the Multnomah County Hospital. No attempts are made herewith to proffer suggestions, its being held that this is rightly the province of the surgeons concerned. It is, nevertheless, an interesting topic for speculation. It is noted that when activity commences in the surgery and the doors are permitted to remain open, the bacterial count rises. The assumption that this increase is due

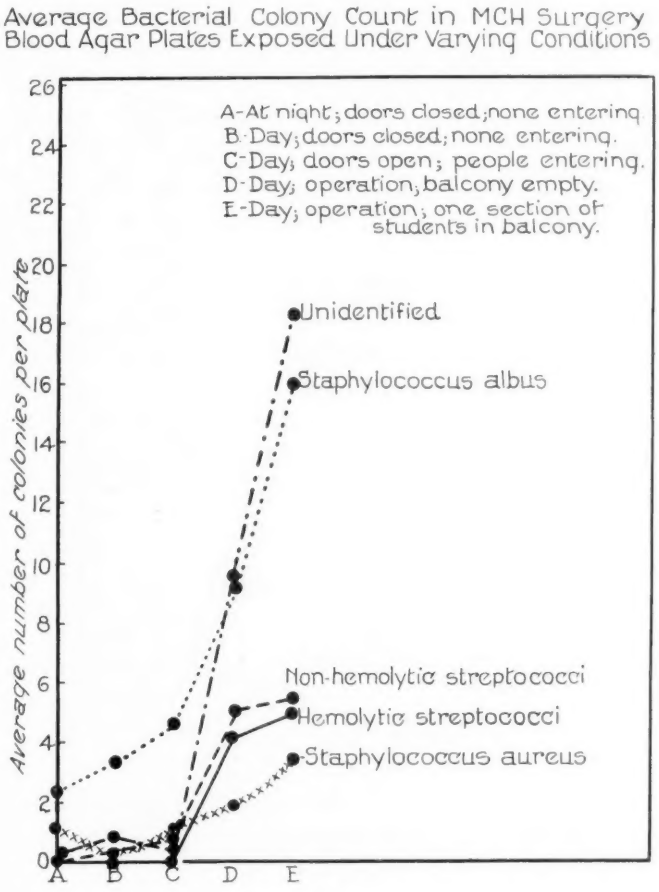

Fig. 3.

in part to the agitation of air currents by passage of the operating room assistants does not appear to be unwarranted. It is possible that some bacteria are carried into the surgery by the footgear. Meleney has suggested that a pair of shoes to be worn exclusively in the surgery be provided by the surgeon and his assistants.

Much variation in masking is noted among the surgeons operating at the Multnomah County Hospital. Some mask only the mouth, allowing the nose to project over the mask. Others include both the nose and the

mouth. Some of those who approach closely to the operating table in the role of spectators (internes and residents) are entirely unmasked and ungowned. There is, it seems, in some instances, a possibility of inadequacy of masking. The sharp increase in bacterial count occurring as soon as students are placed in the balconies is of interest. None of the students are gowned or masked and many of them are found in the balconies in street clothing, surely a possible source of added contamination.

Since the hemolytic streptococci and *Staphylococcus albus* show the greatest proportionate increase after activity commences in the surgery, it appears very probable that the major source of contamination is the nasopharynx. These two organisms are well known inhabitants of the nasopharynx and seem to dominate the bacterial picture as soon as people enter the surgery.

Many methods for reducing the number of bacteria in operating rooms have been suggested, among them being: (1) meticulous gowning and masking of all who enter the surgery and exclusion of all who manifest an upper respiratory infection (Meleney), (2) adequate masking with specially constructed masks (Waters), (3) separating the balconies from the surgery by plate glass partitions (Meleney), (4) irradiation of the air immediately surrounding the operative field (Hart), (5) sterilization of the air in the surgery by filtering through bactericidal solutions (Walbum and Reymann), and (6) providing covers for all exposed sterile surfaces (Foster).

SUMMARY

1. The number of air-borne bacteria in the major surgery of Multnomah County Hospital is roughly proportional to the number of people present in the surgery and to the activity on the floor proper of the surgery.

2. The greatest proportionate rise in incidence is manifested by the streptococci and the *Staphylococcus albus*.

3. The organisms showing this rise are acknowledged to be common inhabitants of the nasopharynx.

4. The estimated number of bacteria falling per hour on a sterile operating field compares unfavorably with similar figures obtained in other hospitals.

The authors wish to thank Dr. H. J. Sears, head of the bacteriology department of the University of Oregon Medical School, for his interest and suggestions.

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OSTEOPSATHYROSIS

With a Report of Three Cases*

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In 1833 Lobestein described a type of bone fragility he had observed among a group of people who suffered multiple fractures and dislocations as a result of slight trauma. He designated this disease "osteopsathyrosis." Since that time many others have described the same bone pathology under the following titles: osteomalacia congenita, rachitis foetalis annularis, malacia myeloplastica, periosteal dysplasia, dystrophia periosteal, osteitis parenchymatosa chronica, micromelia annularis, osteoporosis congenita, fragilitas ossium, and osteogenesis imperfecta.

Although Lobestein drew attention to the abnormal skeletal portion of the body, later writers have pointed with greater force to the blue sclera and other variable characteristics associated with osteopsathyrosis. These variable factors have been studied in their incidence to determine their value in the diagnosis of the disease.

Osteopsathyrosis is described as occurring in three age groups. (1) The *congenital* group are born dead or die soon after birth and are found to have received numerous fractures during delivery. (2) The *infantile* group are apparently normal at birth, but in their first few years experience repeated fractures and dislocations. Vrölick, in 1849, designated this form as fetal rickets. (3) The *juvenile* group do not show the skeletal abnormality until late childhood or about the age of puberty. It is notable that this condition, like many other diseases, shows periods of remission and activity.

The case histories of three patients diagnosed as having osteopsathyrosis are presented. It is to be emphasized that these patients are related, as the family tree indicates.

CASE REPORTS

Case 1.—G. T., a white female, aged 6 years, was admitted to the Memorial Hospital on Jan. 17, 1930. The mother stated that when the child was three weeks old she received a fracture of the left humerus when lifted from her bed. At seven weeks, as she was being turned, she received a fracture of the left femur. Later, a clavicle and both humeri were fractured on separate occasions. By the time she was 6 years old, ten additional fractures had occurred. With the exception of one fracture of the left tibia, these later fractures were equally distributed between the two femurs. After each fracture, rapid union of the fragments took place with the formation of abundant callus.

*This study was aided by a grant from the Valentine Research Fund.
Received for publication, May 20, 1938.

Physical Examination.—At the age of 6 years this girl was 40 inches tall. The skin, hair, and nails were normal. The head was normal in size, shape, and proportion. The eyes were equal in size and shape, and there was a slight azure hue to the sclera. The ears, nose, and mouth were essentially normal. The tongue, mucous membranes, and gums were normal; some dental caries was present. There was no lymphadenopathy. The chest and abdomen were essentially normal. The genitals were normal. Grossly, the extremities were normal except for some slight deformity at the sites of repeated fractures.

Fig. 1.—Photograph of patient (Case 1) at age of 6 years. Note beginning distortion of the left thigh from repeated fractures.

Laboratory Data.—Urine normal. Test for Bence-Jones protein, negative. R.B.C., 5,400,000; W.B.C., 9,000; hemoglobin, 92 per cent. Stool examinations negative for ova, parasites, and fatty bodies. Basal metabolic rate plus 8. Wassermann negative.

BLOOD ANALYSIS		SPINAL FLUID ANALYSIS (MADE SIMULTANEOUSLY WITH BLOOD)
Calcium	11.5	5.2
Phosphorus	4.5	1.9
Total protein	7.7	
Serum albumin	5.9	
Globulin	1.8	

X-ray Examination.—A summary of the x-ray findings in Case 1, extending over a period from the age of 5 weeks to 6 years, is as follows:

There is no miliary granular appearance of the skull vault or flat bones as is characteristic of hyperparathyroidism. There is a generalized "ground glass" density of most of the long bones with slight thinning of the cortices and an irregular trabeculation at their distal ends. The epiphyses at the knees are normal in appearance. The epiphyseal centers of the patellas are just beginning to show.

Old healed fractures of the femurs are present and more recent fractures surrounded by abundant callus are also seen. There is slight anterior and lateral bowing of the left femur.

A flat plate of the genitourinary tract does not reveal evidence of calcium deposits in the region of either kidney. A barium enema demonstrated a normal colon with a competent ileocecal valve.

This series of x-ray studies does not show the effects of scurvy, rickets, or syphilis.

Case 2.—A. T., a white female, aged 19 years, was admitted to the Memorial Hospital on July 4, 1929. She was brought to the hospital unconscious, having a subtrochanteric fracture of the right femur which resulted from an automobile accident. Her recovery from this accident was uneventful. In her past history there had been repeated fractures involving nearly all the bones of the body. X-rays revealed many healed fractures, most of which were in malposition.

Physical Examination.—The woman was obese, dwarfed, and appeared to be much older than 19 years. Her head was normal in size and appearance. The eyes, ears, nose, and mouth were not remarkable. The heart and lungs were normal. The abdomen was pendulous, but no other abnormality was noted. There was marked bowing of both thighs, especially at the hips, but no measurable inequality in length. There was an unusual body contour found to be the result of the distortion of proportions accompanying a marked scoliosis and lordosis of the spine. The thorax was elongated and narrowed and the pelvis was shallow and flattened.

Laboratory Data.—Urine normal. Test for Bence-Jones protein, negative. R.B.C., 4,500,000; W.B.C., 8,000; Wassermann negative.

X-ray Examination.—X-ray plate of the skull shows both the inner and outer tables as well as the diploe of both parietal bones to be thin. This is most prominent near the longitudinal suture.

A plate of the pelvis and upper femurs shows a flattened and deformed pelvic girdle with coxa vara of both femurs. There is a recent fracture through the neck of the right femur and several healed fractures involving the upper portion of both femurs. The cortex of the femur is thin and the medullary cavity wide. The bones all demonstrate an abnormal "ground glass" density.

Note.—This patient was seen again at the age of 22 years, after a fall. X-rays at this time showed the fracture of three years before to be well healed and there were no later fractures.

Case 3.—W. T., a white male, aged 42 years, was admitted to the Memorial Hospital on June 23, 1935. He had received a fracture of the shaft of the right femur when he "turned about on his heel." Inquiry into his past history revealed that this was the fourteenth fracture of the femur he had had, these injuries being distributed equally between the two sides. The first fracture of the femur occurred at the age of 4 years. There had been a lapse of twelve years between the thirteenth and fourteenth fractures, during which time he had led a normal active life as a telegraph operator. During his boyhood, he had received a fracture of the clavicle while playing football.

He was treated by appropriate fixation of the leg in a plaster cast but failed to develop callus at the site of the fracture. A sliding bone graft was placed over the fracture in the hope of promoting healing but this succeeded in bringing about only a fibrous union.

Physical Examination.—The man was poorly developed and dwarfed in appearance. The skin, hair, and nails were normal. The head was unusual in that the frontal bones were high and the mandible and maxilla were very prominent. The calvarium had the contour of an army fatigue hat, suggesting late closure of the

Fig. 2.—Photograph of patient (Case 3) at age of 45 years. Note advanced distortion of the body resulting from repeated fractures.

fontanels or thinning along the suture lines. The eyes, ears, and nose were essentially normal. All teeth were absent and the gums were atrophic. There was a marked lordosis of the vertebral column and the thorax was short and distorted in compensation to the spinal contour. The chest and abdominal organs were apparently normal. The genitals were normal. Grossly, the upper extremities were normal; whereas, beginning with a flattened pelvis, the lower extremities were markedly bowed and distorted.

Laboratory Data.—Urine normal. Test for Bence-Jones protein, negative. R.B.C., 4,200,000; W.B.C., 9,000. Wassermann negative. Basal metabolic rate plus 7.

X-ray Examination.—On x-ray examination there is a destructive change involving the inner and outer tables and diploe of both parietal bones near the longitudinal suture. There is marked scoliosis and lordosis of the spine with corresponding deformity of the chest. There is marked deformity of the pelvis and coxa vara of both femurs with marked inward bowing of both tibia and fibula. At the ends of most of the long bones there is an increased trabeculation associated with a thin cortex and a wide medullary cavity. All the bones of the body show an unusual "ground glass" density. However, there are no definite miliary granular changes in the skull vault as seen in cases of hyperparathyroidism.

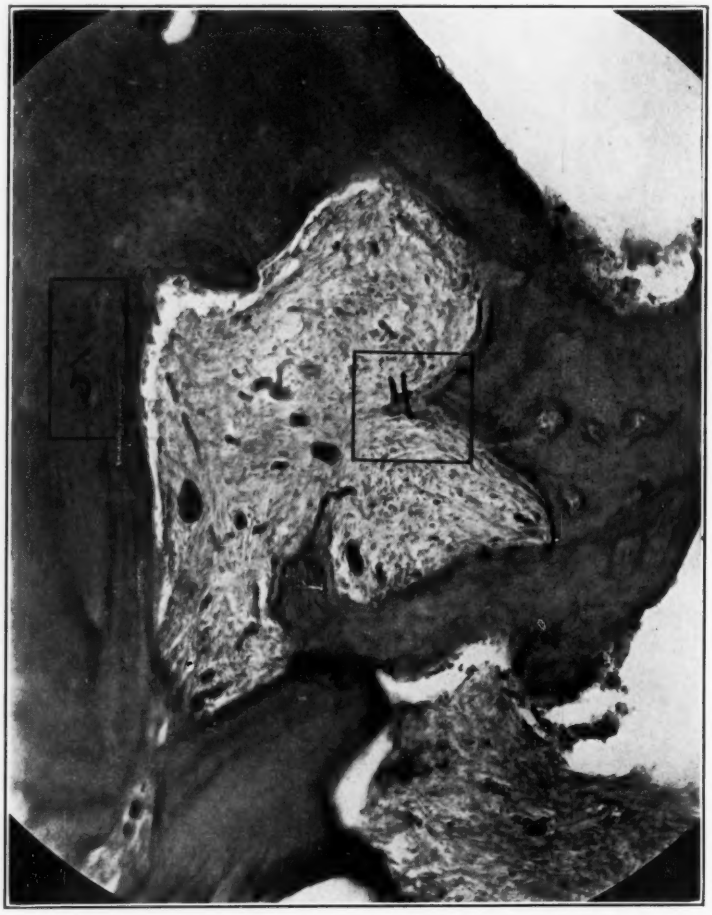

Fig. 3.—Photomicrograph of section of bone removed from the femur (Case 3). This demonstrates the unusual architecture of the bone and the relationship between the fibroblastic stroma and solid bone which form the cortex of the femur. The photomicrograph shows the sites selected for high power photomicrographs shown in Figs. 4 and 5.

An x-ray of the genitourinary tract does not demonstrate any density in the region of the renal pelves.

BIOCHEMISTRY

Biochemical studies involving a large number of determinations on Cases 1 and 3 revealed no remarkable departure from the normal. The average blood chemistry values are given in Table I.

Fig. 4.

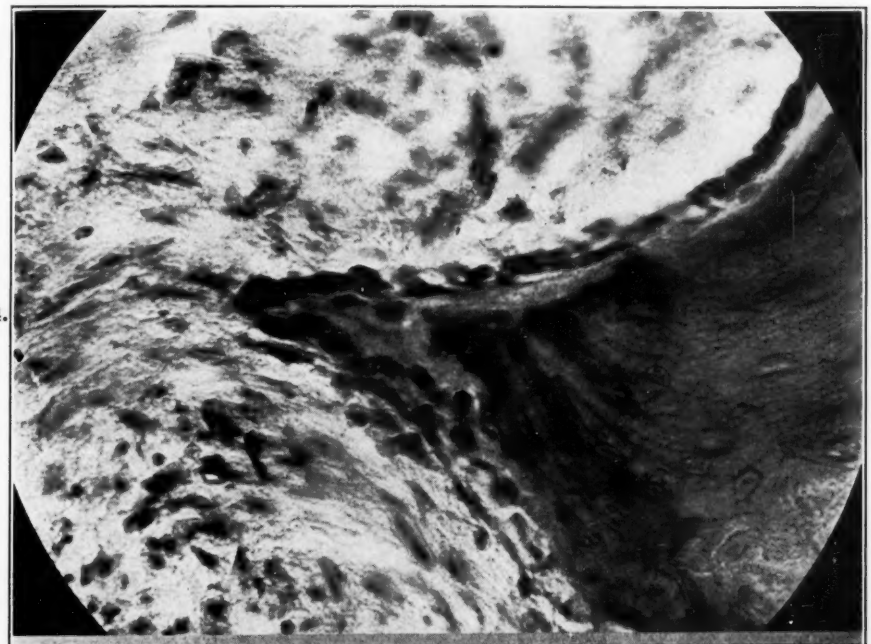

Fig. 5.

Fig. 4.—High power photomicrograph of section of bone from the femur, showing active osteoblasts at the point of contact between the fibroblastic stroma and the solid bone.

Fig. 5.—High power photomicrograph of solid portion of the cortex, showing the abnormal lamellar structure.

TABLE I

BLOOD CHEMISTRY STUDIES

	N.P.N.	SUGAR	CALCIUM	PHOSPHORUS	PHOSPHOTASE*
Case 1	24	.85	11.08	4.5	6.8
Case 3	31	.95	10.5	4.5	6.3

*Bodansky method with a value of 5.4 in the blood of a normal individual.

Calcium and phosphorus retention studies while the patients were on a normal diet were not significant. Only the average values for the whole period in each case are to be considered, since there must always

Fig. 6.—Lateral view of the head of Case 3, showing thin diploe of the skull and failure of closure of anterior fontanel.

be a carry-over from one short period to the next. As the length of periods of observation is increased, greater accuracy of the results is obtainable.

It was demonstrated that there is no difficulty in absorption of calcium and phosphorus from the enteric tract since more than 50 per cent of the phosphorus excretion appeared in the urine. In comparing the values submitted in the Tables II and III, one must resort to tables of the normal for the age of the patient and not check them against each other.

TABLE II*†

CALCIUM AND PHOSPHORUS RETENTION STUDIES, WHILE PATIENT RECEIVED NORMAL DIET

	FIRST PERIOD (2 DAYS)				SECOND PERIOD (2 DAYS)				THIRD PERIOD (2 DAYS)				DAILY AVERAGE RETENTION
	FOOD	URINE	FECES	RETAINED	FOOD	URINE	FECES	RETAINED	FOOD	URINE	FECES	RETAINED	
A. Case 1													
Calcium	1762	147	1130	485	2172	150	1750	272	2259	223	978	1058	302
Phosphorus	2017	1277	671	69	2706	1330	863	513	2832	1221	514	1097	279
B. Case 3													
Calcium	1605	456	1291	−142	2142	340	893	909	1943	328	930	685	242
Phosphorus	2155	1365	775	15	2060	936	636	488	2112	915	608	589	182

TABLE III*

CALCIUM AND PHOSPHORUS RETENTION WITH CALCIUM PHOSPHATE AND COD LIVER OIL FORTIFIED WITH VITAMIN D COMPLEMENT

	FIRST PERIOD (3 DAYS)				SECOND PERIOD (3 DAYS)				THIRD PERIOD (3 DAYS)				DAILY AVERAGE RETENTION
	FOOD	URINE	FECES	RETAINED	FOOD	URINE	FECES	RETAINED	FOOD	URINE	FECES	RETAINED	
Case 1													
Calcium	7299	358	6613	328	6248	309	6002	−37	6443	416	4768	1259	172
Phosphorus	6403	2224	4040	139	5512	1243	3475	794	5130	1952	2622	556	165

*All values are reported in milligrams.

The calcium and phosphorus content of the diet was determined by analysis of identical trays.

†The author wishes to acknowledge the cooperation of Miss Kathryn Heitshu, of the Dietetic Department, and Dr. J. C. Forbes, of the Biochemical Department, in the preparation of these tables.

It is to be noted that, when Case 1 was fed a diet to which calcium phosphate and cod liver oil fortified with vitamin D complement were added, the calcium and phosphorus retention was not increased, but

Fig. 7.—Anteroposterior view of the right femur (Case 3). The thin cortex and wide medullary cavity with the irregular striations are most striking. This x-ray was made shortly after the occurrence of the fracture shown and the rarefication of the bone is not the result of disuse atrophy. A number of healed fractures are also seen in the more proximal portion of the femur.

actually appears to have been decreased. This agrees somewhat with Bookman's suggestion that calcium retention is subnormal in this disease.

The values for blood calcium and phosphorus, as determined in two of the cases presented here, compare favorably with those submitted by

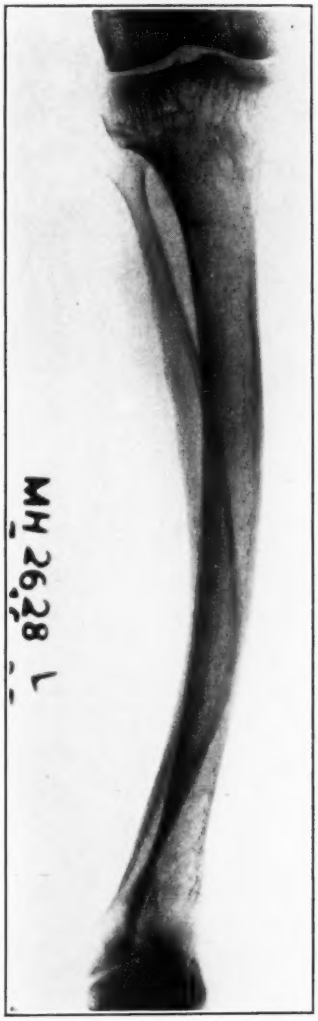

Fig. 8.—Tibia and fibula from Case 3, showing the thin cortex and wide medullary cavity with linear striation of the bone. The bowing is compensatory to the distorted femur.

Gregory and Andersch in the instance of the normal and are in no way related to the values they obtained in the cases of hyperparathyroidism.

The conclusion to be drawn from these chemical studies is that the pathology of osteopsathyrosis is not related to the general calcium and phosphorus metabolism.

PATHOLOGY

Observed at the time of operation, the femur of Case 3 was a yellowish-gray color with a rough irregular surface, the whole appearing more like a bundle of fine spicules than solid bone. The medullary cavity contained a pale yellow fatty material which melted on pressure. There was practically no bleeding from the cut surface of the bone.

According to Wagoner, who studied the amputated leg of an osteopsathyrotic, the fresh specimen was spongy in appearance, the periosteum ragged, the cortex thin and friable, and the marrow soft, edematous, and fatty. The histologic examination showed that the

Fig. 9.—Pelvis and upper femurs of Case 3, showing multiple healed fractures of both femurs. The thin cortex and wide medullary cavity are shown. From the x-ray viewpoint the structure of the femurs and pelvic bones is very similar. The ground-glass density is striking.

periosteum was slightly thickened but in general presented a normal structural appearance. Its outer layer was densely fibrous and its inner portion somewhat more cellular and vascular. The internal coats of a few small arteries were thickened. The osteogenic layer was inconspicuous and appeared unproductive. The cortex did not form a continuous ring but was broken into large and small segments, which showed marked distortion of the Haversian canals and concentric lamellae. Many canals were extraordinarily large and appeared to be filled with marrow substance. In contrast, other canals were small, narrow, and apparently compressed. The bulk of the sections was com-

posed of loosely arranged bony trabeculae which simulated spongy bone and branched throughout the medullary cavity. These trabeculae intermingled with islands of compact bone. Between them were irregular marrow spaces occupied by a delicate stroma containing myxedematous substance in which were a few fat cells, a moderate number of round cells, and many small blood vessels. Closely approximating the islands of compact bone were small groups of multinucleated osteoclasts. Rows of osteoblasts clothed the majority of osseous trabeculae.

The microscopic picture of the bone removed from Case 3 agrees closely with that described by Wagoner. A portion of a section shows what appears to be normal bone surrounded by an osteogenic zone in which the osteoblasts show activity. Adjacent to the spicule of normal appearing bone the structure is that of a rather atrophic fibrous connective tissue. Nowhere is there evidence of the lamellar structure normally seen in cortical bone.

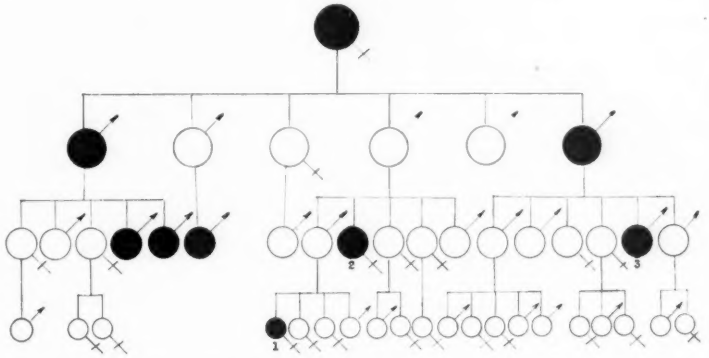

Fig. 10.—Genealogic table. The darkened circles indicate members of the family with fragile bones. Numbers *1*, *2*, and *3* correspond with the cases reported.

DISCUSSION

Practically, it is impossible to trace the transmission of bone fragility as a genetic factor in the human family. Deductions can be made only from evidence as it appears. When more exact knowledge of the influence of endocrine secretion on bone structure is available, deductions will be more accurate. This must have been the thought of Voorhoeve when he described the etiology of osteopsathyrosis as "an inherited inferiority of the mesenchyme." Terry showed evidence of heredity in 9.8 per cent; Griffith, in 26 per cent; and myself in 25 per cent of the cases studied. It has sometimes appeared that osteopsathyrosis is transmitted as a sex-linked factor. Hanson and Griffith have said that the factor for bone fragility may be readily transmitted from the first to the third generation through an unaffected female. Transmission through two unaffected individuals occurred in the family presented here, while other members of the same generation were affected; but

contrary to Hanson and Griffith's experience the unaffected members were the father and grandfather of the child, Case 1. (See family tree for indications, Fig. 10.)

Conrad and Davenport stated that the factor which determines imperfect brittle development of the bones in osteopsathyrosis is a simple dominant one. It is equally facile to show that, if the condition is due to a genetic factor, it may be heterogeneous or a recessive one. At this point it must be recalled that in approximately 75 per cent of the instances, the condition appears idiopathically and that unequivocal cases have appeared in practically every race and nationality. Caucasians predominate but negro cases are not infrequent and a case is reported in a pure American Indian. Peculiarly, the condition seems to be self-limited in that after four generations it vanishes as suddenly as it came.

It appears that other factors besides the gene for bone development must be responsible for this disease.

According to Bronson, in 1900, Eddows associated blue sclera with bone fragility. Since that time all too many cases have been diagnosed osteopsathyrosis by the observance of blue sclera in a patient with a history of one or more fractures. Griffith reviewed 67 cases without mention of blue sclera. In 50 cases of osteopsathyrosis reviewed by myself, blue sclera was found in 72 per cent of the patients. There is a noticeable azure blue color of the sclera of Case 1 in this paper. On the other hand, blue sclera may occur more frequently than has been recorded. Ruggeri quoted Burrows, who described a family of 29 individuals in four generations, 14 of whom had definitely blue sclera, but only 7 had evidence of bone fragility. Ruggeri also quoted Drigthon, who found a family of 14 individuals in four generations, 9 of whom had blue sclera and of these only 5 had fragile bones.

Numerous attempts have been made to explain the source of the blue sclera. Bronson cited Buchanan who in 1908 made a histologic study of eyes with blue sclera. He described the cornea as being three-fifths as thick as normal and the sclera one-third the usual thickness. He found a decrease in the total amount of fibrous tissue of the sclera but no change in the individual fibers. Later, Muir and Patterson stated that in their opinion the blue sclera was of normal thickness, but translucence was increased, allowing the dark choroid layer to be perceptible.

The evidence available does not permit one to admit more than the fact that blue sclera is frequently observed in cases of osteopsathyrosis.

There are also a number of physical abnormalities in addition to blue sclera which have been frequently described as part of this disease. Bronson, Terry, Stewart, Wagoner, and others have pointed out the deafness which is seen in some of these patients. Statistics, however, show an incidence of only 8 per cent. The nature of this deafness has been poorly defined. Fraser stated that it is the result of deposition of

calcium in the middle ear. It has also been suggested that in many instances there is pressure on the eighth nerve somewhere in its course through the thickened irregular temporal bone.

Bronson called attention to the prominent frontal and occipital bones of the osteopsathyrotic skull but definitely contrasted this deformity with the square, box-shaped skull of the rachitic. Thin fontanels frequently persist late in life and the suture lines are prominent. The heads of new bone osteopsathyrotics have been described as "bags of bones with eggshell thickness."

The exophthalmus present in some osteopsathyrotics has no significance other than secondarily to the contour of the skull. It is difficult to establish poor dentition as part of the disease. Many osteopsathyrotics have been said to have infantile genitals, but the cases I have seen showed no such abnormality. However, it is questionable whether or not it is possible for an osteopsathyrotic mother to deliver a living child if her pelvis is of the flattened shallow type.

TREATMENT

The treatment of fracture or dislocation occurring to an osteopsathyrotic is no different from that in a normal person. In most instances healing occurs very rapidly with the formation of abundant callus. Poor reduction and fixation of fractures are the causes of most of the distortion of the bones. So frequently have some of these patients had fractures and dislocations that they learn to treat their own injuries by simple splinting after restoration of the contour of the injured bone.

The administration of calcium, phosphorus, cod liver oil, and various extracts has not produced any effect on the structure or fragility of the osteopsathyrotic bone. Henderson injected thymus gland extract (Hanson) in two cases, but he was not able to demonstrate a change in bone structure. Hanson's thymus extract is being used in the treatment of Case 1 at present.

SUMMARY

Osteopsathyrosis is a disease of the skeleton characterized by marked fragility. It apparently is inherited, although the manner of genetic transmission is not evident.

The x-ray and microscopic pictures of the bones are relatively constant and unlike any other pathologic entity.

There is no biochemical evidence to show that the disease is related to pathologic calcium or phosphorus absorption and retention.

Blue sclera frequently, though not constantly, appears in osteopsathyrosis.

Treatment of fractures of osteopsathyrotics is the same as that for normal individuals. Rapid healing with abundant callus is the rule.

The administration of various drugs, vitamins, and extracts has not been demonstrated to affect the growth or structure of the bone in this disease.

Three cases of osteopsathyrosis treated at the Medical College of Virginia Hospitals are presented.

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RUPTURE OF THE ESOPHAGUS IN A CHILD TWO YEARS OF AGE, WITH RECOVERY

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RUPTURE of the esophagus is usually a fatal accident, death resulting in twenty-four to forty-eight hours from a fulminating mediastinitis and pleuritis. The site of rupture is practically always just above the diaphragm, and, from the published reports, the left lateral wall is more prone to this accident than the right. It has been emphasized by McKenzie[1] that this is the weakest portion of the esophagus.

According to Boerhaave,[2] Baron Wassenau died from a spontaneous rupture of the esophagus in 1724. Spontaneous rupture has been found mainly in adults, but Menne and Moore[3] in 1921 reported such an accident in an infant of 5 months of age, following repeated vomiting. The rupture occurred 1 cm. above the cardia, and there was collapse of the left lung with some spilling of the esophageal contents into the left pleural cavity. Many observers have strongly contended that spontaneous rupture of the esophagus occurred because of preceding inflammatory changes in the wall of the esophagus. Williams and Boyd[4] (1926) concluded from their studies on dogs that this opinion was justified. Brosch[5] concluded that spontaneous rupture of the esophagus would occur following vomiting (which act causes increased intra-esophageal pressure), or, secondarily, from digestion of the esophageal wall by the gastric juice, causing ulceration. Murdoch[6] reported a rupture of the esophagus due to indirect violence; the patient was a child who had been run over by a motorcycle. At post mortem, the rupture was found ¾ inch above the diaphragm, with the left lung collapsed and a small amount of fluid present in the left pleural cavity.

Rupture of the esophagus secondary to direct trauma, such as instrumentation, has been a serious complication which is usually followed by fulminating mediastinitis and death. Perforations, either small or of gradual onset, have been known to recover without surgical intervention. Ballin and Saltzstein[7] in 1922 reported a perforation of the esophagus, resulting in a right-sided empyema. This was treated by drainage, with rib resection, and ultimate recovery occurred.

The following case report is that of a child admitted to the Children's Hospital of Michigan because of difficulty in swallowing:

Received for publication, May 23, 1938.

C. B., white male, 2 years of age, entered the hospital on Aug. 6, 1935, with a history of having been well until July 30, 1935. The swallowing of a foreign body on the date was suspected. It was reported that, previous to admission to the hospital, the child had eaten very little and had vomited repeatedly since the onset of symptoms. Physical examination revealed a well-nourished white male of 2 years, appearing dehydrated. His temperature was normal; pulse, 110; respirations, 24. The fontanels were closed; pupils were equal and reacted to light; ears and nose, negative; tongue, dry; and the throat, slightly injected, but no foreign body was seen. There was no rigidity of the neck, but on examination of the chest, an oc-

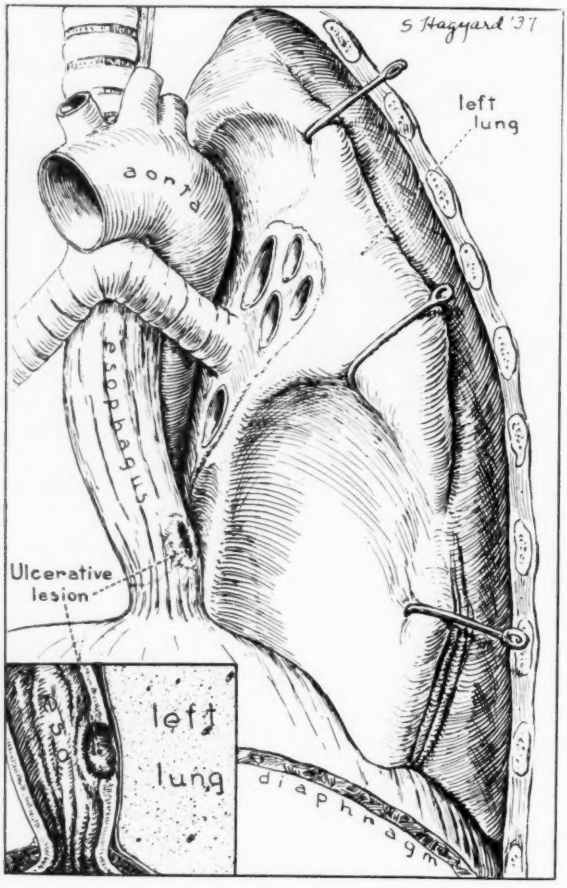

Fig. 1.—The authors' conception of the location and type of rupture of the esophagus.

casional coarse râle was heard over both bases. The heart borders were normal, and no murmurs were heard. The abdomen was soft; liver and spleen were not palpable. A diagnosis of suspected foreign body in the esophagus was made. On Aug. 7, 1935, the patient was esophagoscoped by Dr. A. E. Hammond, who found no evidence of a foreign body in the esophagus, but did see an ulcer approximately 1 by 1 cm. in the left lateral wall, about 1 inch above the cardia. The floor of the ulcer was necrotic and surrounded by a zone of hyperemia. After this examination, the child strained considerably and vomited. Approximately four hours later, there were signs of a left tension pneumothorax, which was aspirated, 400 c.c. of air and 20 c.c. of serosanguineous fluid being removed, with marked relief of the dyspnea.

This finding of tension pneumothorax with fluid in the pleural cavity led us to suspect that the esophageal ulcer had perforated into the left pleural cavity. Fluoroscopic examination at 9:00 P.M. revealed that there was a total collapse of the left upper lobe and 50 per cent collapse of the left lower lobe, with marked displacement of the heart to the right. The child was placed in an oxygen tent with no apparent improvement. Because of the increase in the severity of the tension, pneumothorax, and the inability to control it by aspiration, it was deemed advisable at 11:00 P.M. to institute a closed catheter type of drainage under water. This resulted in marked relief of the dyspnea and cyanosis. Unknowingly, a student

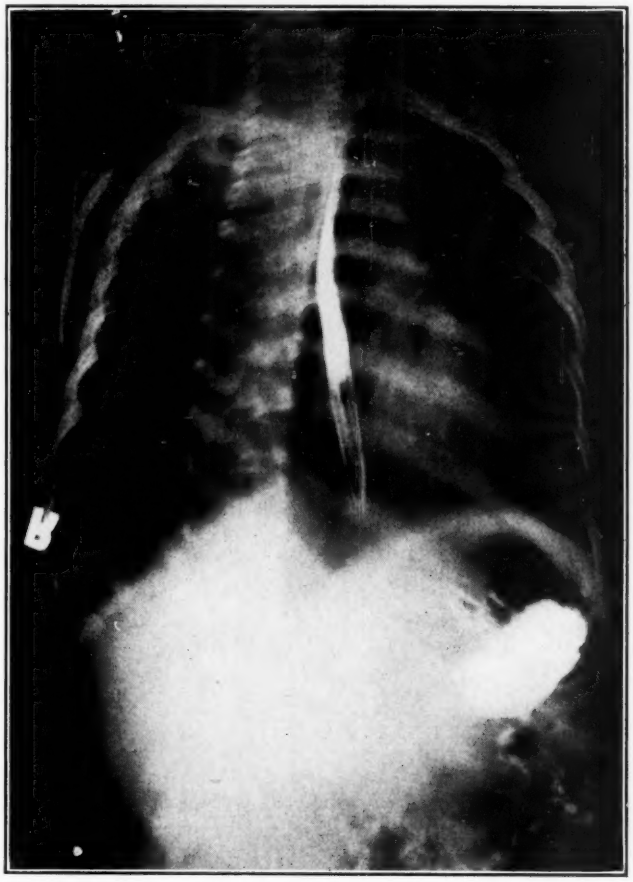

Fig. 2.—Small amount of barium given and x-ray taken approximately 2 months after rupture of lower end of esophagus.

nurse gave the child milk to drink, and immediately the milk appeared in the drainage tube which had been placed in the left pleural cavity. This definitely proved that there was a perforation of the esophagus into the left pleural cavity. A Levine tube was inserted into the stomach for feeding purposes, putting the esophagus at rest. Fluoroscopic examination and an x-ray film Aug. 8, 1935, showed the drainage tube in the left pleural space, with no indication of free air or fluid in the left pleural cavity. The rectal temperature varied from 102° to 104° for two days following the rupture of the esophagus. Aug. 15, 1935, x-ray examination revealed the lung almost completely re-expanded. The Levine tube was used for feeding purposes until Aug. 20, 1935, when fluids were given by mouth. The fluid which drained

through the catheter showed no growth on culture. Aug. 26, 1935, the tube was removed from the pleural cavity and the child was discharged from the hospital four days later. Check-up examination and fluoroscopic study Sept. 30, 1935, when a small quantity of barium suspended in milk was given, showed that there was no interruption in the downward progress of the barium into the stomach. The lumen of the esophagus was reported small, but there was no abnormality in the contour of this structure at the site of the reported previous rupture. May 24, 1937, or twenty-one months after the rupture, there were no symptoms referable to the esophagus or thoracic organs. Fluoroscopic examination after the administration of barium revealed no evidence of obstruction or stricture in the esophagus.

McKenzie[1] has emphasized that the weakest point in the esophagus is just above the diaphragm. This anatomic weakness predisposes to rupture in this portion of the esophagus. The case herein reported had an ulcerative lesion present which made rupture easy following vomiting and straining. Boyd and Williams,[4] as quoted by Gott,[8] urged early surgical drainage of the pleural cavity. Gott[8] (1933) reported four cases of spontaneous rupture of the esophagus, one of which had a thoracotomy performed on the tenth day, and expired on the seventeenth day of illness.

We believe that rupture of the esophagus must be considered in all cases of sudden collapse following instrumentation of the esophagus, indirect violence to the thorax or abdomen, or repeated vomiting. If this lesion is suspected, the esophagus should be put at rest by carefully inserting a Levine tube for feeding purposes and early drainage of the pleural cavity instituted by the closed catheter method. Radical surgery by immediate thoracotomy and suture has been reported as futile.

CONCLUSIONS

1. A rupture of the esophagus in a child 2 years of age, with recovery, is reported.

2. Preceding local disease of the esophagus predisposes to spontaneous rupture following vomiting.

3. Patients in whom prostration, dyspnea, substernal pain, subcutaneous emphysema of the neck occurs should suggest esophageal rupture.

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Recent Advances in Surgery

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PROGRESS IN SURGERY OF THE AUTONOMIC NERVOUS SYSTEM

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THE intrusion of neurosurgery into the field of cardiovascular and other forms of visceral disease has been so dependent on contemporary investigations of the fundamental laboratory sciences that the compiler of a review of recent surgical progress must include much of the current work in neuroanatomy and physiology. For this reason, no apology is due for incorporating a large proportion of papers which at first glance might seem to be more suitable to a journal devoted to special laboratory investigation. Similar reviews of advances in surgery of the autonomic nervous system have appeared annually in the *New England Journal of Medicine* since 1930;[1] the reader who is interested in a summary of the principal papers which have appeared in preceding years may refer to them.

ANATOMY AND PHYSIOLOGY

Recent work of far-reaching importance has given an insight into the way the involuntary nervous system adapts the body to emotional and physical changes. Investigation of the activity of higher centers in the cerebral cortex has been carried out by the Department of Physiology at Yale and has been summarized by Fulton.[2] He points out that the activity of structures, such as the heart, blood vessels, gastrointestinal tract, and secretory organs, are immediately subject to integrations occurring at the cortical level. As early as 1876, Hughlings Jackson[3] had suspected that visceral functions must have extensive representation in the cerebral cortex, since these functions are almost invariably disturbed during epileptic seizures emanating from this level. Little was done to verify Jackson's hypothesis until the last few years. The systematic investigation of Fulton and his colleagues by stimulation and ablation of cortical areas in the cat, dog, and monkey has demonstrated that the principal areas in which autonomic responses have been obtained on stimulation overlap those giving somatic reactions. It is clear that the frontal lobes, and in particular those areas known in the cyto-

Received for publication, October 3, 1938.

architectural maps as areas 4 and 6, are regions in which integrations occur simultaneously in both the autonomic and somatic spheres. Thus, when a vigorous volitional movement occurs, the muscles involved may be supplied automatically with an increased amount of blood, the reaction occurring as a direct innervation from the cortex, rather than as a secondary response to local accumulation of metabolites. The studies of Hoff and Green[4, 5] on curarized animals indicate that, when the premotor cortex is stimulated, the muscles affected show an increased volume which can only be due to an increased flow of blood through them. The overlying skin exhibits little change. When limb volume increases, that of a viscus like the kidney is diminished. Furthermore, during vigorous skeletal movement the cortex through its gut representation is capable of directly shifting the blood from the visceral to the muscular bed and inhibiting the activity of the gut. On the basis of these experiments, it is concluded that a mechanism exists by which the requirements for increased blood supply in muscles brought into action by the motor cortex may be anticipated.

These newer disclosures concerning the relation of the cerebral cortex to the autonomic nervous system give an adequate physiologic basis for the long-recognized relationship between mental states and visceral processes. It is thus clear that the load which is placed upon the circulatory system may be determined in large measure by the mental state of the individual. In emphasizing this point, Fulton aptly states that "the heart and circulation may be worked just as hard, and just as much as a detriment to the body as a whole, from an arm chair—or perhaps I should say a swivel chair—as from a rower's seat." As Cannon[6] has pointed out, many disturbances of the thyroid, cardiovascular, and gastrointestinal functions undoubtedly have a similar basis. In résumé, it seems highly probable that with more thorough analysis all autonomic functions will be found to be to a certain extent under cortical control.

The work of many physiologists and clinicians, which has been summarized in previous reviews of progress in this field,[1] has resulted in a corresponding advance in knowledge of the functions of the hypothalamus. As stated by Papez,[7] not only areas 4 and 6 on the lateral surface of the cerebral hemispheres, but also the medial cortex in the region of the gyrus cinguli and the hippocampal formation have important connections with the hypothalamus and the anterior thalamic nuclei. It is these related areas which probably elaborate the functions of central emotion and give rise to emotional expression. This integration meets the physiologic requirements proposed by Cannon[6] and Bard[8] in respect to the theory of corticodiencephalic processes. It is also in agreement with the observations of Dandy[9] that the seat of consciousness is located somewhere near the midline, between the limits set by the corpus callosum and the basal structures of the brain.

The evidence that the hypothalamus contains the central ganglia of the autonomic nervous system is well summarized in Ranson's Harvey Lecture.[10] He shows that the hypothalamus exerts its important regulatory functions through two channels. By way of the hypothalamico-hypophyseal tract it controls secretion of the antidiuretic hormone from the posterior lobe of the hypophysis and thereby the rate of elimination of water through the kidneys. The interruption of this tract results in atrophy of the pars nervosa and causes diabetes insipidus. Secondarily, through its connections with the rest of the brain and spinal cord, the hypothalamus regulates visceromotor activity. When released from cortical inhibition, as in Bard's decorticate cats, or when stimulated electrically in Ranson's and in Beattie's[11] experiments, the hyperactivity of these centers causes dilation of the pupils, bristling of the hair, elevation of the blood pressure, and on the somatic side rapid respiration, struggling, clawing, and biting; in other words, the picture of emotional excitement. Conversely, damage to the hypothalamus produces emotional stolidity and somnolence. Working at least in part through the sympathetic nervous system, the hypothalamus serves also as a thermostat in the regulation of body temperature. After anterior lesions, cats tend to develop postoperative hyperthermia and are unable to protect themselves against rising external temperatures. After posterior lesions the animals run subnormal temperatures and are easily chilled. Possibly through injury to the descending pathway from the anterior hypothalamus, these animals are also unable to protect themselves against overheating and become truly poikilothermous.

Similar alterations in the temperature regulation of human beings have been observed in tumors which infiltrate the hypothalamus. Davison and Friedman[12] report a child with destruction of the hypothalamic nuclei by an infiltrating neuroblastoma who showed extreme temperature fluctuations from 103° to 93°. Other cases cited by Davison and Friedman have been described in recent years, and Peet and Kahn[13] have observed extraordinary vasomotor changes in the extremities in patients with lesions in this area. Indeed, it is highly probable that states of chronic vasoconstriction (Raynaud's disease) and hyperhidrosis ultimately will be connected with abnormal hypothalamic function.

A final point of interest in connection with stimulation of the hypothalamus is the report of Magoun, Ranson, and Hetherington[14] that large amounts of adrenaline and sympathin are liberated in the blood stream under these circumstances. This is of direct clinical importance in relation to the residual ability of smooth muscle to contract after denervation.

In a monograph entitled *Autonomic Neuro-Effector Systems* Cannon and Rosenblueth[15] take up the discovery of acetyl choline by Loewi and of sympathin by Cannon and his co-workers. The authors trace the development of this new field of neurophysiology and its implications.

Of greatest practical concern to the surgeon is the finding that smooth muscle is rendered abnormally sensitive to circulating adrenaline and sympathin, a fact first observed by Meltzer and Auer[16] in 1904 and then nearly forgotten until the recent revival of interest in chemical transmission of the nerve impulse. The experiments which established this fundamental principle have been reviewed in Cannon's George E. Brown Memorial Lecture[17] before the American Heart Association and in the recent review of "Advances in Sympathectomy for Peripheral Vascular Disease" by Peyton and Titrud[18] which recently appeared in this JOURNAL, as well as in previous articles in this series.[1d-f] Suffice it to point out here that the sensitization phenomenon is found in all smooth muscle which is under the control of adrenergic nerve impulses, and that its effects have been measured on the denervated heart and nictitating membrane,[15] as well as on the peripheral vascular tree. McCloskey, Co Tui, Mulholland, and Wright,[19] have found that its action may become so intense as to produce necrosis of the skin when adrenaline is injected intracutaneously in the lower forelimb of a dog after stellate ganglionectomy. The impossibility of producing a chronic flaccid form of paralysis in smooth muscle is explained in large part by this sensitization to chemical mediators which are liberated in the circulating blood whenever the sympathetic nervous system is called into activity. Whether there is another gland of internal secretion (for example, the posterior lobe of the pituitary or the adrenal cortex) which supplies a chemical agent that might account in part for the restoration of tone in sympathectomized smooth muscle is not known. Since Govaerts'[20] experiments, it is also established that the peripheral sympathetic ganglia, after their central connections have been cut, develop a rhythmic discharge of their own. Cannon[17] feels that restoration of tone after denervation must result in part from an intrinsic property of smooth muscle itself, because it is found in the completely sympathectomized animal.

OPERATIVE METHODS

A better understanding of the puzzling discrepancy in results following sympathetic ganglionectomy of the upper and lower extremities has resulted from the explanation of the peculiar behavior of smooth muscle after denervation. It has been pointed out in the preceding section that there is always a considerable restoration of tone in any form of denervated smooth muscle. This has been found to vary in degree with the level at which the central or peripheral pathway is cut. Whereas lumbar ganglionectomy has given consistent relief of vasospasm in patients with Raynaud's disease of the lower extremity, careful follow-up examinations after resection of the inferior cervical, first and second thoracic ganglia frequently have shown a striking degree of residual vasospasm on exposure to cold or excitement, even in patients with

indubitable evidence of complete regional sympathectomy. The fact that smooth muscle of the arterial walls becomes some ten times more sensitive to circulating adrenaline and sympathin after degeneration of its postganglionic fibers* and but three times more sensitive if only preganglionic fibers are cut has been most clearly demonstrated by Ascroft.[21] Previous, less carefully controlled experiments by Grant,[22] and by White, Okelberry, and Whitelaw[23] have been in close agreement. White[24] had first pointed out that, inasmuch as the synapses between the pre- and postganglionic neurones occur in the inferior cervical and upper two or three thoracic ganglia in the case of the hand and in the lowest lumbar and upper two sacral ganglia in the case of the foot, resection of the second and third lumbar ganglia is a preganglionic sympathectomy; whereas, cervicothoracic ganglionectomy causes degeneration of the entire postganglionic network to the arm. This concept that an excessive sensitization to chemical mediators in the circulating blood is responsible for the poor results of cervicothoracic sympathectomy has been upheld by Cannon,[17] Telford,[25] Learmonth,[26] and Ascroft.[21] Ascroft has gone one stage further and shown in monkeys that the results of a previously satisfactory lumbar ganglionectomy can be vitiated if the sacral ganglia are subsequently resected.

This peculiar reaction of the denervated vascular wall to adrenaline, sympathin, and possibly to other chemical substances in the circulating blood therefore must be taken into account. Normal vessels react slightly to adrenaline, but after any form of sympathectomy this effect becomes exaggerated. This can be noted within twenty-four hours of denervation and reaches its maximum between the tenth and fourteenth days. The degree of sensitization then persists at about this level unless regeneration occurs. If this takes place, the heightened response subsides (Simeone[27]). Fortunately sensitization is a clinical handicap only after postganglionic sympathectomy. If care is taken to interrupt the peripheral vasoconstrictor pathway only in its preganglionic component, a most satisfactory vasodilation results and is maintained.

Standardized technical procedures are now available which fulfil these physiologic requirements:

Lumbar Ganglionectomy.—For the relief of vasospasm in uncomplicated Raynaud's disease of the lower extremity, resection of the second and third lumbar ganglia can be counted on to produce effective and lasting vasodilation. Since the fourth lumbar ganglion gives off a few postganglionic fibers to the sciatic nerve, it is best to leave it in place.

*For those who do not understand the terms pre- and postganglionic, it may be stated that there are two sets of fibers in the peripheral sympathetic pathway. The upper or preganglionic fibers have their cells of origin in the lateral horn of gray matter in the thoracic and upper two lumbar segments of the spinal cord. Their axones pass by way of the anterior nerve roots and white rami communicantes to the paravertebral sympathetic chains, in which they run cranially or caudally for varying distances before forming synapses with other ganglion cells. From the latter, lower or postganglionic axones arise, which pass to the brachial and lumbosacral plexuses in the gray rami communicantes. These lower axones run in the mixed nerves to the peripheral vascular tree.

In patients with Buerger's disease, where it is desirable to produce the maximum vasodilation in the upper leg as well, Fontaine, Houot, and Dos Santos[28] have given clear-cut experimental evidence that the resection should be carried upwards to include the first lumbar ganglion. When this is done, the male patient should be warned that he will become sterile through loss of the power of ejaculation.

Lumbar ganglionectomy is best carried out by the posterior extraperitoneal approach. Excellent modifications of Royle's original muscle-splitting incision have been worked out by a number of surgeons and are described by Pearl.[29] These permit exposure of the entire length of the lumbar chain from its first to fourth ganglia. Even if a bilateral resection is to be undertaken, the consensus of recent opinion has favored a two-stage extraperitoneal approach over the older transperitoneal incision because of its increased safety and the smoother convalescence.

Thoracic Sympathectomy for Preganglionic Denervation of the Upper Extremity.—Two operative procedures recently have been advocated which have given most satisfactory results in the hands of their originators. That of Smithwick[30] consists of a posterior paravertebral approach with resection of the medial portion of the third rib and corresponding part of the transverse process. The sympathetic trunk is divided caudal to its third thoracic ganglion and its cephalic end swung up and buried in the intercostal muscles. As a few more rostral vasomotor fibers emerge over the second and third intercostal nerves, these also must be divided. The secret of success in this operation depends on a careful central dissection of these nerves down to the intervertebral foramina and on making sufficient traction so that they can be cut off central to their point of division into anterior and posterior roots. If this point is neglected, a few rami to the brachial plexus are usually left intact and some residual vasoconstriction and sweating from central impulses is almost certain to persist.

The operation devised by Telford[25] and now widely used in England utilizes the cervical approach of Gask and Ross.[31] After cutting across the anterior scalene muscle, the apical pleura is freed from the sides of the vertebrae as far down as the third rib. The rami communicantes which connect the second and third thoracic ganglia to the corresponding intercostal nerves are then divided and the sympathetic trunk cut below the third thoracic ganglion. The crucial step here is to swing the cephalic portion of the chain up as far as possible and to bury its end in one of the cervical muscles. Preganglionic fibers have an extraordinary capacity for regeneration and the two divided ends of the chain must be widely separated. In his earliest cases Telford neglected to carry out this step, with the result that Simmons and Sheehan[32] observed a gradual return of sweating and vasoconstriction in three patients within six months. In addition to the diminished degree of residual vasoconstrictor

tone which results from the preganglionic operation, another advantage is that the ocular fibers are not interrupted and thereby the patient is spared the disfiguring ptosis, pupillary constriction, and enophthalmos of a Horner's syndrome.

In summarizing these important findings in the field of peripheral vascular disease, it seems fair to conclude that there is no occasion to question the general physiologic principle of the sensitization phenomenon which appears with the denervation of all smooth muscle. The increased sensitization which follows postganglionic denervation also has been fully corroborated by many different investigators. Whether the present surgical methods for accomplishing this type of sympathectomy are fully satisfactory, time alone can tell.

EFFECTS OF SYMPATHECTOMY ON CIRCULATION

Carotid Sinus Syndrome.—Heymans and Brouha[33] have contributed a very complete review of the reflex control of circulation by the carotid sinus and the cardioaortic nerves. One important function of this mechanism is to assure an adequate flow of blood through the vital centers of the brain, which are so easily damaged by lack of oxygen. These investigators not only have found that vasopressor and depressor reflexes originate from these plexuses in the vascular walls, but also they describe other accessory vascular reflex centers in the splanchnic area. All of these give off impulses to the higher vasomotor centers when they are stimulated by alterations in either the blood pressure or the chemical content of the circulating blood.

A further clinical study of the carotid sinus and other sensitive areas in the periphery which may set up visceral reflexes has been contributed by Ferris, Capps, and Weiss.[34] These authors find that surgical denervation of the carotid sinus does not alter visceral symptoms in patients with vegetative neuroses, and that in these states many other parts of the body may become conditioned as reflex centers so that their stimulation may produce a great variety of symptoms and signs. Such areas as the eyeball, pharynx, larynx, bronchus, pleura, esophagus, and arteriovenous aneurysms have been cited as examples of sensory stations which under abnormal conditions can influence efferent portions of the autonomic nervous system in the same manner as the hypersensitive carotid sinus. With the realization that other peripheral areas may send afferent stimuli to the central ganglia of the autonomic nervous system, it becomes obvious that the carotid sinus alone cannot play a major role in regulating the tonus of the autonomic nervous system. Mechanical stimulation of an irritable sinus, however, can produce three well-recognized syndromes: (1) periods of bradycardia or asystole; (2) transient falls in blood pressure; and (3) syncopal attacks and epileptiform convulsions.

Weiss, Capps, Ferris, and Munro[35] have shown that in the first two conditions medication with atropine and ephedrine is sufficient to prevent vagal and vasodilator reflexes. Only the third variety of abnormal response may require surgical intervention. Freedberg and Sloan[36] have described four such cases, in two of which lasting freedom from the convulsive seizures was secured by denervation of the irritable zone at the carotid bifurcation. In the other two the attacks could be stopped by novocainization of this area, but no operation was carried out. Robinson's[37] recent finding that the tendency to this form of syncope and convulsions can be relieved by benzedrine sulfate deserves a careful trial and may save this group of cases from the need of surgical denervation.

Raynaud's Disease.—In planning sympathectomy for patients with vasospasm the surgeon must never forget that the term Raynaud's syndrome includes a number of pathologic conditions. These were listed by Lewis and Pickering and have been discussed in detail by Lewis[38] in his monograph on vascular disorders of the limbs:

"1. Intermittent spasm of digital arteries, without complications.

"2. Intermittent spasm of digital arteries, with local nutritional changes.

"3. Intermittent spasm of digital arteries, with generalized scleroderma.

"4. Raynaud's phenomenon arising out of local injury, including the use of vibrating tools.

"5. Bilateral gangrene of digits in the young, and with infection; this condition is regarded as probably the result of thrombotic closure of vessels.

"6. Bilateral gangrene with haemoglobinuria from cold. . . .

"7. Bilateral gangrene of digits in the elderly, in which closure is shown to be thrombotic, but preceded by disease of the small arteries.

"8. Thrombo-angiitis obliterans associated with Raynaud's phenomenon.

"9. Cervical rib or crutch pressure causing Raynaud's phenomenon or gangrene . . . by thrombotic and embolic processes."

Learmonth,[26] in his excellent review of surgery of the sympathetic system, has stated that the possibility of treatment by sympathectomy arises in any of these conditions except 6, 7, and 9. He believes "that operation should be recommended in groups 1 and 2 if the attacks continue to be troublesome after the patient has placed herself in the best possible environmental conditions. In group 3 operation may be offered, but with less assurance of relief, for the vessels are too confined by new fibrous tissue. In group 4 change of occupation is imperative, and operation should not be offered or undertaken until this has been arranged for. Group 5 should be offered operation, with a view to limit-

ing the process and encouraging repair." In Group 8 sympathectomy is being found to be of increasing value.

Lewis[38] has always contended that Raynaud's disease is due to a primary fault in the smooth muscle of the digital arterioles with local susceptibility to cold. This is undoubtedly true in the cases of Group 2, where nutritional changes have developed local fibrosis and often actual endarteritic changes in the digital arteries. Whether Lewis's contention is correct that vasoconstrictor activity is not increased to an abnormal degree in cases under Group 1 remains to be proved. Opinion in this country has been that in the early uncomplicated stage of the disease sympathetic tone is abnormally high. Evidence in support of this has been summarized by Adson.[39]

The controversy between those who believe that poor results of sympathectomy in the upper extremity are due to incomplete denervation and those who feel that an unphysiologic operation (postganglionic denervation) is the principal factor has not been fully settled. Certainly other factors play a role, such as the tendency for the disease to be more severe in the hand than in the foot, and the fact that vasoconstrictor tone is greater in the lower extremity (Doupe, Robertson, and Carmichael[40]).

The results of preganglionic sympathectomy in Raynaud's disease of the upper extremity, when properly performed, speak strongly in favor of the second of the two theories mentioned in the preceding paragraph. Perhaps the most convincing argument to bring out the importance of interrupting vasoconstrictor impulses in the upper preganglionic axone has been presented by De Takats.[41] He reports observations on four patients suffering from Raynaud's disease, in whom preganglionic sympathectomy had been performed on one side and postganglionic on the other. The contrast between the two sides was striking. Out of four preganglionic sections, his results were excellent in three and only fair

TABLE I

RESULTS OF SYMPATHECTOMY IN RAYNAUD'S DISEASE

SIXTEEN SYMPATHECTOMIES ON SIX PATIENTS*

CASE NO.	NAME	DURATION	OPERATION		RESULT		REMARKS
1	W. F.	3 yr.	Preganglionic	1	Fair	1	
			Postganglionic	1	Failure	1	
2	N. S.	8 yr.	Postganglionic	2	Failure	2	Residual sweating
3	A. M.	6 yr.	Preganglionic	1	Excellent	3	
			Postganglionic	1	Failure	1	Brachial neuritis, 6 wk.
			Lumbar	2			
4	A. J.	1 yr.	Preganglionic	1	Excellent	1	
			Postganglionic	1	Fair	1	
5	P. Z.	10 yr.	Preganglionic	1	Excellent	1	Brachial neuritis, 4 mo.
			Postganglionic	1	Fair	1	
6	K. S.	8 yr.	Postganglionic	2	Fair	2	
			Lumbar	2	Excellent	2	

*Patients in this series have been followed through at least one winter, three of them through two winters. By fair result is meant definite improvement, fewer or shorter attacks, but not complete relief.

in one in whom residual sweating indicated that denervation had not been complete. The comparatively poor results of the older cervicothoracic operation are so well brought out in the table of De Takats' operative results that it is here reproduced as Table I.

Smithwick, who has probably had the largest operative experience in this field, is at present reinvestigating a series of 70 cases, some of which have been followed for three years. These are being studied by testing the absence of any rise in temperature after novocain injection of the ulnar nerve, paralysis of the sweat glands, and freedom from sudden alterations in the psychogalvanic responses and in blood flow through the fingers measured with the photoelectric cell. With a very few exceptions, mostly in the earliest cases where the second and third intercostals were not divided within the intervertebral foramina and where sympathetic denervation was incomplete from the start, the early excellent results have been maintained.

Other authorities who have commented on the importance of limiting resection to the preganglionic sympathetic fibers, in order to avoid the extreme sensitization of the denervated vascular wall to adrenaline and sympathin, include Telford[25] and Learmonth.[26] Up to date, however, the follow-up statistics of the new methods which have been advocated to carry out a preganglionic denervation of the upper extremity have been too meager to evaluate their ultimate effectiveness.

Thromboangiitis Obliterans.—Learmonth,[26] in reviewing a series of cases with this disease, has classified them in certain groups:

"1. An early group, usually (and wrongly) labelled Raynaud's disease in the male.

"2. A group in which the disease is very slowly progressive, so slowly that it may appear to reach a certain stage, after which it does not progress. Thus cases are not rare in which claudication has been the only symptom for many (e.g., fifteen) years.

"3. A group in which the progress is episodic, the episodes corresponding to waves of thrombosis.

"4. A group in which the disease progresses rapidly to massive gangrene."

It is in Groups 1 and 2 that sympathectomy is indicated in the presence of a suitable vasodilator response to heating or novocain block. The operation "may obviate or at worst delay amputation; or it may allow a below-knee rather than an above-knee amputation." Both Learmonth[26] and Telford[42] have come to the conclusion that "when the disease affects the lower extremities it is unwise to consider the problem as unilateral, the so-called sound leg often being 'sound' only because it has been spared by the earlier appearance of claudication in the other." Therefore, operation should usually be bilateral.

Gask and Ross,[31] in the second edition of their textbook, state that after sympathetic ganglionectomy:

About 40 per cent of patients remain comfortable and are able to return to full work.

About 25 per cent have relief of pain and their circulation is much improved, but insufficiently to permit a return to work.

About 25 per cent show no appreciable improvement.

About 10 per cent require amputation.

In addition, De Takats[41] and Telford[42] have reported follow-up observations in 32 patients. In Telford's 22 cases, all of which have been followed from three to six years, 16 remain well and the disease has made no further progress. The other 6 display some degree of deterioration and further evidences of the disease. Two have recurrent attacks of phlebitis and 4 have required some form of amputation. From these Telford concludes that the benefit from operation is due entirely to dilation of the collateral bed. In De Takats' opinion claudication will not improve following sympathectomy unless subsequent vascular exercises are capable of enlarging a very limited vascular bed. Also, the operation cannot be expected to ameliorate the intractable rest pain which accompanies a fixed vascular bed. Here the most useful surgical procedure is the crushing of the sensory nerves to the foot, as originally described by Smithwick and White.[43]

No outstanding recent advances have been reported in connection with sympathectomy for various other conditions. The improvement of circulation in the cold, blue, partially paralyzed legs which may result from poliomyelitis is of considerable value in carefully selected cases (De Takats[41]). Scleroderma, when it is limited to the extremities with accompanying vasospasm, may show limited improvement.[26, 31] The recent method of combining parathyroidectomy with sympathectomy is outside the scope of this review. In the treatment of the intractable forms of hyperhydrosis where the excessive sweating is limited to the palms and soles, sympathetic denervation of the sweat glands continues to be the only effective treatment. Excellent charts of the regions over which the sweat glands are paralyzed have been published by Roth.[44] Finally, in the treatment of the rheumatoid form of arthritis, which was widely advocated a few years ago, more careful investigation of the effects of sympathectomy has shown that improvement can be expected in only a very limited group of cases (Learmonth[45]).

Hypertension.—Arterial hypertension is a clinical sign, not a disease. While its causes are varied, experience demonstrates that the patients in whom the cause is known and can be dealt with surgically are extremely rare. One of the most interesting varieties is caused by tumors of the adrenal medulla which secrete large quantities of adrenaline and thereby produce paroxysmal attacks of hypertension. Accounts of these in the literature are not new, but Beer, King, and Prinzmetal[46] have been able to demonstrate the excessive amount of adrenaline in the circulat-

ing blood of a patient during such crises by perfusing a sample of plasma through the denervated arteries of a rabbit's ear. This caused constriction of the sensitized vessels. The pressor effect disappeared after removal of the tumor and was not present on perfusion with normal plasma. Another variety of hypertension due to an abnormality of the endocrine glands is seen in the basophile adenomas of the pituitary. No satisfactory surgical solution has been advanced for this condition. The same applies to hypertension in cardiovascular and renal disease, with the rare exception of nephrectomy when a single kidney is diseased.

The treatment of essential hypertension by various operative procedures represents the newest excursion of surgery in therapy. This subject is at present in a state of evolution and it is highly important to follow the work of both the experimenter and the clinician. Very little encouragement has been given the surgeon from the laboratory. It is known that blood volume and viscosity, as well as cardiac output, are normal in this disease. The resistance to flow of blood through the arterioles is increased, but the question remains whether the reason for this is due to increased vasoconstrictor tone or to an abnormal pressor substance circulating in the blood. Page[47] reports that as yet there is no direct evidence of any pressor substance in the blood, urine, or spinal fluid. Nonetheless, it has been proved conclusively that hypertension in the Goldblatt dog with partial compression of the renal arteries is not neurogenic. Freeman and Page[48] have been unable to produce any fall of blood pressure in these animals after complete sympathectomy and cardiac denervation. Furthermore, hypertension has been produced by Glenn, Child, and Heuer,[49] as well as by Blalock and Levy[50] through constriction of the artery of a single transplanted kidney devoid of a nerve supply. The only procedure which has been found to reduce this type of hypertension is bilateral adrenalectomy.[47] This effect is not due to lack of medulliadrenal secretion, but to the cortex, and fits in with the hypothesis of Page[47] that cortical secretion is concerned with the maintenance of the vascular system in a state of reactivity. When this secretion is lacking, it is possible that the vessels can no longer respond to chemical pressor stimuli. Although no abnormal pressor substances have been demonstrated in the blood of these animals, physiologic evidence points indubitably to their presence. If the mechanism of essential hypertension is due to a fault in the blood supply of the kidney, it is possible that this may be improved by sympathectomy.

A true neurogenic form of hypertension has been produced in dogs by Heymans and Bouckaert[51] by resecting the carotid sinus and cardioaortic depressor nerves. In such an animal normal blood pressure can be restored by total sympathectomy, but no lasting reduction results from resecting the splanchnic nerves and the lumbar sympathetic chains.

While experimental evidence has offered little hope of surgical success in the attempt to reduce essential hypertension, operative results in human beings have been distinctly more encouraging. The unsatisfactory results of medical treatment and the high mortality of the disease make any surgical attempt at relief justifiable, provided it carries a low risk. Experimental animals cannot be submitted to the cortical emotional drive (see above) to which the pressor centers in the human hypothalamus are exposed, and it is well known that patients with essential hypertension exhibit an abnormal pressor response to such stimuli as emotional excitement or sudden exposure to cold. Allen and Adson[52] have shown that these rises in blood pressure may be greatly reduced in patients after sympathectomy. These authors report a series of 85 patients treated by splanchnic and lumbar sympathectomy at the Mayo Clinic. Fifty-five per cent responded by a good fall in blood pressure (an excellent reduction to practically normal values in 25 per cent), while about 70 per cent showed favorable subjective and clinical improvement. There were no operative deaths in the entire series. The operation itself does not disable, although anhydrosis of the lower extremities and loss of ejaculation and probably of fertility in the male result. In the favorable cases the heart may decrease in size, inverted T-waves in the electrocardiogram may become upright, retinitis and spasm of the retinal arteries may diminish or disappear, albuminuria may decrease and renal function improve. But even after the most careful possible selection of cases, blood pressure was not materially reduced in 45 per cent.

Freyberg and Peet[53] have discussed the effects of bilateral splanchnicectomy in patients with essential hypertension. In general they have observed that changes in the kidneys are associated with changes in blood pressure. In those patients who had a significant and maintained fall, urinary abnormalities decreased or disappeared. Their observations indicate that in primary hypertension renal efficiency is not dependent on the higher blood pressure, as has been commonly thought since Traube, in 1856, first postulated the Compensatory Theory of the cause of the elevated blood pressure. The authors believe that the impairment of renal function is caused by vascular constriction and that if the constriction is relieved by splanchnicectomy the activity of the kidneys is improved.

Page and Heuer[54] have studied the cardiovascular and renal changes in 9 patients after thoracic splanchnicectomy and interruption of the thoracic sympathetic chain. Three of these suffered from the malignant form of the disease and cannot fairly be included. In the others they were unable to detect any improvement in cardiac or renal efficiency. The reduction in arterial pressure was marked, but within six months it had returned to the preoperative level in all patients. In the 6

patients with the more benign form of the disease, the reduction of headaches, fatigability, nervousness, and irritability was striking, although in 3 improvement lasted less than a year; in the 3 with malignant hypertension it was but transient. The therapeutic results in this small but representative group of patients are certainly not encouraging.

The results of the same operation in the hands of Peet at Ann Arbor and Smithwick[30] at the Massachusetts General Hospital have been very similar to those after the subdiaphragmatic resection of the splanchnic nerves and upper lumbar ganglia at the Mayo Clinic. But in spite of these more favorable reports, it has not yet been established that the expected duration of life in hypertensive patients can be prolonged, even if the blood pressure is reduced. More accurate methods for selecting cases favorable for sympathectomy and longer observation of patients who have been subjected to this procedure are the two most important points required to place the surgical treatment of essential hypertension on a sound basis.

VISCEROMOTOR CONTROL OF AUTONOMIC NERVOUS SYSTEM

Megacolon.—The term Hirschsprung's disease signifies a congenital dilatation of the colon of neurogenic origin and serves to distinguish this condition from the acquired form which is caused by chronic obstruction or by faulty habits of defecation. Experience with improvements in the technique of denervation suggests that neurosurgical methods may entirely supplant the old and dangerous procedure of colectomy in cases where there is no mechanical obstruction. The act of defecation is accomplished by parasympathetic nerve impulses which arise from the sacral segments of the spinal cord as stretch reflexes. They increase the tone of the detrusor muscle and relax the internal sphincter. In opposition to the sacral autonomic fibers, the sympathetic outflow from the lowest thoracic and upper lumbar segments of the spinal cord causes inhibition of detrusor activity and a tightening of the involuntary sphincters. In a recent monograph Passler[55*] has assembled information concerning the origin and treatment of megacolon from many angles.

Adson[56] reviews the various forms of sympathectomy which have been advocated. He argues that surgical intervention should depend on an evaluation of the degree of the disease and emphasizes the importance of selecting a method of denervation that will include sufficient sympathetic inhibitor fibers to re-establish a balance of the neuromuscular mechanism. In the milder group of cases, where the disease is limited to the lower portion of the descending colon, left lumbar ganglionectomy (the operation of Wade and Royle) alone is usually sufficient. Bilateral

*This monograph has been highly recommended in the reviews of new books, but it has been impossible to obtain a copy from any library to date.

resection of the lumbar chains, as advocated by Judd and Adson, has been found to be still more effective. The operation of Rankin and Learmonth, which consists of the removal of the inferior mesenteric and superior hypogastric plexuses, has failed to give satisfactory results in the more advanced cases of Hirschsprung's disease. None of these operations are effective in relieving symptoms when the ascending and right half of the transverse colon are involved, nor in the atonic dilatation which accompanies the acquired form of the disease. For these conditions Adson has been resecting the splanchnic nerves together with the first and second lumbar ganglia bilaterally through posterior incisions carried out in two stages. This operation, which interrupts the sympathetic inhibitor fibers to the small intestine and ileocecal valve as well as the supply to the colon, promises to give far more effective results and should be given a wide trial. It must be remembered, however, that it will produce sterility in the male through a loss of the power of ejaculation. The same objection applies to operations which interfere with the nerves descending along the aorta and the superior hypogastric plexus.

SYMPATHECTOMY FOR VISCERAL PAIN

Learmonth[26] has summarized current anatomic knowledge of the viscerosensory nerves as follows: "Pain fibers from the viscera have the same morphological arrangement as somatic afferent fibers; that is, they are the axons of cells in the posterior root ganglia. Some of these fibers utilize sympathetic pathways to reach the spinal cord. They leave a viscus by its individual nerve-strands, usually traverse peripheral sympathetic ganglia, and the majority reach the paravertebral sympathetic chains by way of the pelvic splanchnic nerves (pelvic viscera), thoracic splanchnic nerves (abdominal viscera), and cervical splanchnic nerves (thoracic viscera). After entering the paravertebral chains they may run cranially (or caudally) for varying distances; finally they traverse white rami communicantes, and thence pass by way of the posterior root ganglia and the posterior nerve-roots to the spinal cord. . . . Although the mechanism of production of visceral pain cannot be regarded as finally settled, the majority of observers are agreed that it arises as the result of tension in the viscus, whether this is the result of distension, of inability to evacuate its contents, or of inco-ordinated muscular contraction."

While distention seems to be the principal cause of pain in colic of the hollow viscera, another mechanism must account for arterial pain. Moore and Greenberg[57] have studied the mechanism of pain in coronary occlusion. Having previously found that lactic acid causes striking pain responses when injected into a coronary artery, they determined the acidity of the blood obtained from the veins of the functioning heart

at varying intervals after ligation of both coronary arteries. The coronary venous blood showed a marked lowering of pH and the lactic acid content was greatly increased. In three animals the pH fell below 6.6 and in one it reached 6.4. Definite pain responses can be elicited by intra-arterial injections as weakly acid as pH 6.3, and it is certain that there would be a greater concentration of acid metabolites in the capillaries and tissue spaces of the laboring muscle in actual coronary infarction than could have been detected in the blood of the coronary sinus during the brief period of these experiments. This is clear-cut evidence that the pain of coronary disease is due to an irritation of the sensory nerve endings in the heart by acid metabolites, just as is the case with skeletal muscle during periods of intermittent claudication.

Interesting observations have been made by Leriche[59] on pain produced by direct stimulation of the central end of the splanchnic nerve in two patients undergoing splanchnicectomy under spinal anesthesia. Each complained of severe pain in the chest; in one instance it was referred to the heart, in the other to the lung on the ipsolateral side.

Ochsner and De Bakey,[59] in their review of the surgical treatment of coronary disease, give statistics on the results of the various methods of cardiosensory denervation. Excellent anatomic diagrams are included to show the arrangement of the cardiac nerves and the importance of interrupting the direct thoracic as well as the cervical fibers. Their statistics show that when cervicothoracic sympathectomy was used there was rather a large number of failures because these operations fail to interrupt the accessory afferent fibers in the direct thoracic rami. As the authors state: "The ideal procedure should be resection of the upper four or five thoracic sympathetic ganglia. In this way all fibers carrying sensory impulses from the heart would be interrupted. However, such a procedure in patients who are admittedly poor risks would undoubtedly carry an unjustifiably high mortality."* In the statistics compiled by Ochsner and De Bakey, about 80 per cent of the 68 patients subjected to paravertebral alcohol injection were relieved of severe anginal attacks which had not responded to medical treatment, with a mortality rate of 1.5 per cent. A certain number of failures are unavoidable, due to the technical difficulty of blocking all of the cardiac rami in thick-chested individuals. Under these circumstances recourse may be had to posterior root section with an almost certain expectation of success, provided the patient is in sufficiently good condition to be subjected to so radical a procedure.

Another successful application of sympathectomy of pain of visceral origin is seen in presacral neurectomy in obstinate cases of dysmenorrhea. Cotte,[60] the originator of this operation, reports a series of 300

*In my own series of four patients where the upper three thoracic ganglia had been resected, relief was complete except for slight residual arm pain in one instance. In four unpublished cases of other surgeons where the upper five thoracic posterior spinal roots have been cut, the pain has been consistently relieved.

cases treated in this way. This operation will effect a cure of rebellious cases of idiopathic dysmenorrhea which have been refractory to all other forms of therapy. Cotte feels that in certain cases reported in the literature failure has been due either to incomplete resection or to the fact that the patients were poorly selected for operation. The operation will not relieve ovarian pain, which travels in the plexuses along the ovarian vessels. There have been no abdominal complications or difficulty with the sphincters. More than 50 patients have had consecutive pregnancies without accident.

CONCLUSIONS

During the course of the eight years in which these reviews of advances in sympathetic neurosurgery have been written, there has been a noticeable restriction of its applications. Its use in epilepsy, for which the first sympathectomy was done, has long since been discarded. So has the attempt to reduce spasticity, undertaken by Hunter and Royle, as a by-product of which the modern operations for Raynaud's disease and megacolon have developed. Sympathectomy has been found to be of little or no value in various conditions for which it has been enthusiastically advocated, such as bronchial asthma, retinitis pigmentosa, atypical forms of facial neuralgia, and in the relief of joint symptoms in rheumatoid arthritis.

On the other hand, a great deal has been learned in the last few years about the integration and control of the involuntary nervous system by the higher centers in the cortex and hypothalamus, and also through such peripheral sensitive areas as the carotid sinus. Likewise knowledge of the anatomic arrangement of the visceral plexuses has become much more exact and has explained the cause of failure in the older operations for angina pectoris and megacolon. A more complete understanding of the physiologic behavior of denervated smooth muscle is leading to better handling of the problems connected with peripheral vascular disease. Interest is now focused on the surgical treatment of essential hypertension, where the value of sympathectomy must still be regarded as *sub judice*. With the exception of this condition, clinical experience has now led to a fairly exact appreciation of what sympathetic neurosurgery has to offer towards the correction of abnormal visceral function and the relief of visceral pain.

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Review of Recent Meetings

FIFTY-SECOND ANNUAL MEETING OF THE AMERICAN ORTHOPAEDIC ASSOCIATION, MAY 2-4, 1938

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THE FIFTY-SECOND Annual Meeting of the American Orthopaedic Association was held at the Ambassador Hotel, Atlantic City, New Jersey, on May 2, 3, and 4, 1938, under the presidency of **Frederick C. Kidner,** Detroit, Mich. This meeting was held in conjunction with the Fiftieth Congress of American Physicians and Surgeons, since the American Orthopaedic Association is one of the component organizations.

J. A. Freiberg, Cincinnati, Ohio, spoke on the scalenus anticus muscle and its relation to shoulder and arm pain. He stated that spasm or contracture of the scalenus anticus muscle may cause pressure on the subclavian artery and brachial roots and may produce combined vascular and nervous symptoms. He enumerated many common lesions of the shoulder girdle and cervical spine, such as calcification of the supraspinatus tendon, arthritis of the shoulder or the cervical spine, and others which are frequently complicated by spasm of the scalenus anticus muscle. He stated that brachial neuritis is usually a sign of the scalenus anticus muscle and that brachial neuritis is usually a sign of scalene spasm or contracture, rather than a true clinical entity. Concerning treatment, the author felt that two factors must be considered, the etiologic factor, or primary lesion, and the degree of vascular and neurologic changes which have occurred. Briefly, Freiberg believes that the initial therapy should be directed to the lesion which is causing the scalene spasm. Combined with this therapy some form of simple balanced traction applied to the arm has relieved the symptoms in several cases. Postural variants, such as the long neck, flat chest type, predispose to the scalene syndrome. Therefore, in some cases postural therapy is indicated. If the symptoms are severe and associated with vascular or neurological or combined changes, tenotomy of the anterior scalenus muscle should be done as advocated by Ochsner.

Eugene B. Mumford, Indianapolis, Ind., read a paper entitled **Osteoporosis.** He did not discuss the theories of osteoporosis, but reported a series of cases treated by x-ray therapy. The treatment consisted of daily doses of 150 to 175 x-ray hours to the affected part for four days. This light dosage, according to Mumford, did not in any case produce any evidence of skin irritation. The dosage could be repeated at the end of three or four weeks.

Paul C. Colonna, Oklahoma City, Okla., presented a paper entitled **An Arthroplastic Procedure for Congenital Dislocation in Children.** Colonna stated that an arthroplastic procedure for congenital dislocation of the hip has been emphasized by him for the past eight years, and this presentation reviewed the technique of

Received for publication, July 15, 1938.

the procedure and stressed the limitation of the operation. The details of the second procedure as originally described in 1932 have remained practically unchanged. The first stage consists of a preliminary stretching of the soft tissues about the hip continued until the head is pulled down opposite the site of the original acetabulum. In the author's hands this has been most satisfactorily accomplished by using skin or skeletal traction while immobilizing the opposite hip in a plaster spica. In the older cases a previous subperiosteal stripping of the gluteal muscles about the crest of the ilium unquestionably has helped in obtaining soft tissue relaxation.

The second stage consists in preserving the hour-glass portion of the capsule covering the head. The redundant capsule about the old acetabulum has been removed and a new acetabulum made with a large curette after which the capsule covered head is placed deeply within the reconstructed acetabulum and the wound closed in layers. After four weeks the plaster spica has been completely removed and active and passive motion started in an overhead swinging apparatus. No weight bearing is allowed for three months following the second stage.

Colonna stresses the fact that the most satisfactory results have been obtained in the unilateral case under 10 years of age and he described the procedure in a movie film showing a number of patients presenting a normal range of hip motion.

Frank R. Ober, Boston, Mass., read a paper on **Discoid Cartilage and Trigger Knee, Pathology and Treatment.** He reported three cases which were diagnosed at the Boston Children's Hospital and one adult private case of the writer's. The treatment for such disability, according to Ober, is that the cartilage must be removed; if this is not done, the mechanical irritation of the cartilage, which is becoming enlarged from the cyst formation, is likely to injure the articular surfaces and produce arthritic changes.

Philip Wilson, New York, N. Y., read a paper entitled **Popliteal Cyst (Baker's Cyst), Its Etiology, Pathology, and Treatment.** He stated that in the majority of cases he found a composite bursa present, consisting of the so-called gastrocnemius and the semimembranosus bursae. This lay under the inner head of the gastrocnemius muscle and under the semi-membranosus tendon, extending posteriorly between these two muscles and finally lying quite superficially under the deep popliteal fascia. The author's conclusion was that popliteal cysts are in the great majority of instances inflammations of the gastrocnemiosemimembranosus bursa and probably the result of traumatic irritation of the cursal sac. The treatment recommended is complete surgical removal.

An afternoon Clinic on Tuesday, May 3, was given over to a **Symposium on bone tumors,** made up of **James Ewing,** New York, N. Y.; **Robert D. Schrock,** Omaha, Neb.; **Bradley L. Coley,** New York, N. Y.; and **Henry W. Meyerding,** Rochester, Minn. **Dr. Coley** discussed **Giant Cell Tumor of the Bone.**

Giant Cell Tumor of Bone, by **Bradley L. Coley** and **Norman L. Higinbotham,** New York, N. Y. After an historical survey, Coley and Higinbotham reported on a series of 124 cases of giant cell tumor of bone from their services at the Memorial Hospital and the Hospital for Ruptured and Crippled. The average age at the onset of the disease was 29.7 years; 45 per cent of the patients were females and 55 per cent were males. As to the bone involved, the distal end of the femur, the proximal end of the tibia, and the distal end of the radius, accounted for 75 of the 124 cases. In the differential diagnosis there were many bone lesions that had to be distinguished from the giant cell tumor, among them:

simple bone cyst, von Recklinghausen's disease, central chondroma or chondromyxoma, medullary osteolytic osteogenic sarcoma, and, rarely, plasma-cell myeloma.

Coley and Higinbotham recognized the two conservative methods of treating the giant cell tumor; namely, surgical extirpation by curettage or resection, and radiation by radium pack or roentgen ray. They reserved radical surgery, i.e., amputation, for frankly malignant cases or for exceptional ones in which the tumor was situated in the lower end of the extremity where an artificial limb assured a proper functional result. They did not feel that the question was finally settled as to whether radiation or surgery was the better method, and the wide divergence of opinion would seem to indicate that neither extreme view was correct. Their experience had led them to adopt surgery for lesions that were accessible, particularly those that were in the region of the knee joint, and radiation for lesions that were inaccessible, particularly those in the spine, pelvis, or neck of the femur. They warned against the use of radiation and surgery in the same case.

The technique of treatment by surgery and by radiation is separately described. In closing they presented tables listing the results of treatment, as follows:

TABLE OF RESULTS OF TREATMENT OF 124 CASES OF GIANT CELL TUMOR

TREATMENT	CASES	EXCELLENT RESULT	GOOD RESULT	POOR RESULT	UNCLASSIFIED
Surgery alone	35	17 or 57%	6 or 20%	7 or 23%	5
Radiation alone	53	14 or 34%	12 or 30%	15 or 36%	12
Combined (surgery and radiation)	34	7 or 22%	8 or 25%	17 or 53%	2
Coley's toxins alone	2	1 or 50%	1 or 50%		

			TYPE OF PREVIOUS TREATMENT		
			SURGERY	RADIATION	COMBINED
Amputations, 26 cases:		For infection	2	0	2
Primary amputation	2 cases	For radiation changes	0	4	2
Secondary amputation	24 cases	For malignant transformation	1	0	6
		For failure to control disease	2	3	2
		Total	5	7	12
		Percentage of followed cases	17%	17%	37%

SUMMARY OF RESULTS OF TREATMENT IN 124 CASES OF GIANT CELL TUMOR AT MEMORIAL HOSPITAL AND HOSPITAL FOR RUPTURED AND CRIPPLED

		CASES
Alive and under observation		83
Lost to follow-up		19
Dead:		22
Causes related to bone tumor	14	
Causes unrelated	4	
Cause unknown	4	
Total		124

The conclusions reached by Coley and Higinbotham were: (1) The giant cell tumor is usually benign but malignant forms are encountered and may result from the transformation of a tumor that is histologically benign at the outset.

(2) Surgical extirpation (curettage, resection, and, on rare occasions, amputation) and radiation have been established as successful methods of treatment. Surgery is preferable for accessible tumors, and radiation for inaccessible or extremely advanced tumors. (3) Caution should be exercised when using roentgen ray without histologic confirmation of the diagnosis, for one may be dealing with an osteolytic sarcoma which bears a close resemblance, roentgenographically, to the giant cell tumor. (4) Radiation should not be used in conjunction with surgery. Each method should bear the full responsibility of its employment in the individual case. (5) Roentgen ray in large doses destroys the regenerative powers of the bone; in small doses it may fail to arrest the disease; therefore, the exact dosage for the individual case is a matter of profound judgment or of fortuitous circumstance. (6) Inexpert radiation is probably less hazardous than surgery in the hands of one unfamiliar with technical operative details. Loss of limb may ultimately result in either instance. (7) Surgical attack should envisage thorough removal of all tumor tissue through adequate exposure, careful wound closure without packing or drainage, and primary wound healing. (8) Protection during the regenerative phase is essential regardless of the treatment employed, for a pathologic fracture usually spells functional impairment and a painful neighboring joint.

Gilbert E. Haggart, Boston, Mass., discussed **True Idiopathic Sciatica** wherein he presented the method of treatment for sciatic pain of unknown origin. He used a method of three procedures in some, a combination of two, but in most instances three: (1) Perineural injection of the sciatic nerve with novocain (1 per cent); (2) traction to the affected extremity; and (3) in an increasing number of cases, manipulation of the low back under intravenous pentothal anesthesia. After carrying out these procedures all patients are instructed in muscle exercises, particularly designed to develop the low back muscles and those of the gluteal group. On becoming ambulatory, posture training is also given as indicated.

Frank N. Potts, Buffalo, N. Y., discussed **Aseptic Necrosis of the Head of the Femur, Following Traumatic Dislocation.** He reported five cases of aseptic necrosis of the head of the femur following traumatic dislocation. In none of these cases were fractures present. The dislocations were reduced in four cases within forty-eight hours and in the one case, within two weeks; no great difficulty was encountered in reduction. Four of the five cases were the result of automobile accidents. There was a long period of well-being, followed by pain, limp, and restricted motion in the affected hip. Radiographic examination showed what Potts believed to be an aseptic necrosis, with a resultant arthritis in the hip. Microscopic study of sections of bone removed showed a thinning of the articular cartilage and break down in the subchondral bone, allowing for the altered shape of the hip joint; there was some attempt to repair by invasion.

These patients, stated Potts, were seriously disabled with a progressively destructive lesion, involving the head of the femur. The diagnosis was readily made on suspicion and with the x-ray findings. With knowledge of this condition, it becomes increasingly difficult to offer a good prognosis in the dislocated hip. While statistics are rather scarce, it would seem from inquiry that this might not be an unusual condition. If the diagnosis was made early enough it is possible that keeping the person from bearing weight might have a beneficent effect, in that it would lessen the chance for alteration in the shape of the head of the femur. It might also be considered that drilling through the head and the neck might bring about some improved blood supply. Undoubtedly, the original pathology is a destruction of the capital blood supply as it goes through the capsular vessels

and ligamentum teres. The treatment rests between an arthroplasty and an arthrodesis; Potts favors the latter procedure. The choice would depend upon the amount of destruction of the head.

Arthur Steindler, Iowa City, Ia., discussed **Nonparalytic Scoliosis, Principles of Treatment and Nonoperative Management.** Steindler stated that first, it is shown that the compensation, that is the realignment of the body over the trunk, is a necessary prerequisite for the success of both conservative and operative treatment. This is documented by long range observation and the statistical curves of these observations show definitely that cases which had an adequate compensatory treatment before fusion, held after the fusion was done, whereas cases in which compensation treatment was not carried out, or could not be carried out to a satisfactory degree, did not hold, but they relapsed after a short initial period of apparent success. The statistical curve of those cases that were secured by conservative treatment and in which adequate compensation was accomplished prior to fusion, did hold during periods of long range observations where the others did not. It appeared, however, from Steindler's observations, that too many cases were selected for conservative treatment. All in the late end results showed by the permanency of the correction that only about 40 per cent of the cases that he saw were suitable to conservative treatment alone.

Steindler believes that the reason for the rather low percentage of long range successes in conservatively treated cases is due to the difficulties of correcting the curve itself, aside from the compensation realignment. Consequently, he goes a step farther in correction of the curve. Steindler believes from x-ray observations that the keynote to the correction of the curve is the derotation. This derotation of the curve is carried out with specially designed equipment, both in the sitting and lying positions. It is to be expected that a higher percentage of permanent control of scoliotic spines, both in the conservative and operative treatment, will result from this added feature of the correction of the curve itself. The correction of the lateral curve is carried out both by active and passive exercises and by the corrective cast methods after Risser. Steindler's total material embraces 1,600 cases. **Drs. W. R. Hamsa** and **Cooper,** Iowa City, Ia., collaborated in this study.

The Presidential address, **Progress of Orthopedic Surgery in America,** was given by Frederick C. Kidner. **T. P. McMurray,** of Liverpool, who was the Association's guest, read a paper entitled **Osteoarthritis of the Hip.**

Book Reviews

Pediatric Surgery. By Edward C. Brenner. Cloth. Pp. 843, with 203 illustrations. Philadelphia, 1938, Lea and Febiger. $10.

With the exception of fractures, all of the surgical specialties pertaining to pediatric surgery are included in this volume. Six topics or specialties, including anesthesia, blood transfusion, cleft lip and palate, thoracic surgery, genito-urinary surgery, and neurosurgery, are very ably contributed by colleagues of the author. The purpose of the book is described in the preface as being of a practical nature and not encyclopedic or compendial in type. Apparently, for that reason the author has entirely omitted a bibliography. To many readers this feature will be considered unfortunate. In the author's endeavor to be concise and practical he resorts to an attitude of dogmatism in numerous sections which would be properly tempered by a generous number of references.

The endeavor to include a bounteous supply of clinical facts has been very efficiently executed. On numerous occasions statements are made which would not meet with complete approval by many surgeons. For example, certainly not all surgeons are as thoroughly convinced of the value of intraspinal tetanus antitoxin as the author is.

From the clinical standpoint, embryological and anatomical data have been very adequately covered. The short introduction outlining the various features in which children differ from adults is very useful and appropriate. Congenital malformations which make up such an important part of pediatric surgery are considered in various parts of the book. Congenital malformations of the rectum are very thoroughly and efficiently described, but the anomalies of the small intestine, which are of so much importance from the standpoint of the infant's life because of the frequency of obstruction, particularly at the duodenum and terminal ileum, are mentioned only very briefly. Other inconsistencies in the amount of space devoted to various subjects are apparent. For example, acute cervical lymphadenitis is such a frequent and serious disease in children that more than one and one-half pages would appear to have been appropriate.

In the preface the author states that he wishes to avoid mentioning procedures, etc., which are of doubtful value. The use of Hartmann's combined solution is not discussed. There are undoubtedly many pediatricians and surgeons who believe this to be a very useful and efficient agent, and to be an effective buffer, particularly in acidosis. In the section on shock, gum acacia is not mentioned as a substitute for plasma when blood is not immediately available. Many surgeons would probably consider gum acacia to be past the experimental stage and of definite therapeutic value.

Certain sections of the book are especially well written and contain a thorough, concise discussion of all the informative data desired. This may be said particularly of the section on acute osteomyelitis, thoracic surgery, and genitourinary surgery.

The reviewer hoped to find a more thorough discussion of postoperative feeding, particularly in such conditions as pyloric stenosis where serious vomiting may ensue as the result of improper or too large feedings, and be corrected so promptly by

changing the feeding or lessening the quantity for 24 to 48 hours. The postoperative administration of fluids, etc., is discussed rather briefly, but the author obviously relies very strongly on the pediatrician to take care of these phases of therapy.

As stated previously, the book contains an enormous amount of practical data so useful and necessary in the surgical care of infants and children. For that reason, it will be a very valuable addition to the library of the pediatric surgeon.

Quelques Vérités Premières (ou soi-disant telles) en Chirurgie Abdominale. By H. Mondor. Pp. 96. Paris, 1937, Masson & Cie. 24 fr.

In 1881 or thereabouts the late Mr. E. Hurry Fenwick, Surgeon to and Lecturer on Clinical Surgery at the London Hospital, gathered together a number of accepted precepts from well-known standard treatises of surgery for the use of his dressers and senior students, to supplement the theoretical knowledge which they had acquired. This compilation proved so popular that it was printed under the title of *Golden Rules of Surgical Practice* and ran through six editions, with additions and changes from Mr. Fenwick's own pen. In 1906 a new book, entitled *Golden Rules of Surgery*, based on the foregoing but with many additions from the rich fund of his own personal experience, was issued by the late A. C. Bernays, of St. Louis, including his original observations on such matters as fees, professional methods, scientific contributions, and economic questions of interest to surgeons.

The volume now under consideration *(Some Fundamental Truths in Abdominal Surgery)* is constructed much on the same plan, except that it is confined solely to the scientific aspect and makes no note of collateral issues. It is one of ten volumes, all made up of paragraphic aphorisms; each volume is by a different writer, under the general editorship of L. Ombrédanne and N. Fiessinger, who state in their preface that while aware that much of the current knowledge of today ultimately will be discarded, there is much that is so definitely established that it can be accepted with finality. It is on this latter type of information that these books are made up, and they cover practically the entire field of modern surgery. All of the authors are members of the Faculty of Medicine of Paris; in the present instance the author is Assistant Professor of Pathological Surgery, as well as Surgeon to the Hôpital Bichat.

He has assembled his material into three major groups. Group A covers the abdominal wall, the stomach, duodenum and pancreas, with subsections on contusions and wounds of the abdomen, traumatic rupture of the spleen, thoraco-abdominal wounds, fibroma of the abdominal wall, strangulated hernia, ulcer of the lesser curvature, ulcer of the duodenum, peptic ulcer of the stomach, cancer of the stomach, benign tumors of the stomach, hour-glass stomach, stenosis of the pylorus and duodenum, perforated ulcers, subphrenic abscess, acute phlegmonous gastritis, acute occlusion of the duodenum, pancreatic cyst, sarcoma of the pancreas, chronic pancreatitis, cancer of the pancreas and acute hemorrhagic pancreatitis. Group B deals with the intestines and peritoneum, giving consideration to acute intestinal obstruction, intussusception, strictures of the small intestine, gallstone obstruction, volvulus of the cecum and pelvic colon, gangrene of the intestine, disease of Meckel's diverticulum, mesenteric cysts, acute and chronic appendicitis, inflammatory tumors of the right iliac fossa, cancer of the cecum, iliocecal tuberculosis, acute peritonitides, typhoid perforations, streptococcus, pneumococcus, gonococcus, and tuberculous peritonitis, cancer of the colon, ulcerative colitis, polyadenoma of the

large intestine, anal fissure, peri-anal abscess and fistula, stricture of the rectum, and cancer of the rectum. The third section is given up to the major problems of gynecology, including tubal pregnancy, complicated abortions, the inflammatory, malignant, and benign conditions involving the uterus and adnexa, etc.

This somewhat extended review of the contents has been given to emphasize the scope and importance of this work. It is full of rich meat, beautifully—even dramatically—written but with the utmost economy of words. It deserves to be translated, for it would make a worthy addition to the literature of any language.

The Practice of Urology. By Leon Herman. Cloth. Pp. 923, with 504 illustrations. Philadelphia, 1938, W. B. Saunders Company. $10.

The Practice of Urology by Leon Herman, Professor of Urology at the Pennsylvania Post-Graduate School of Medicine, is a very satisfactory textbook. It is logically arranged and presented, and admirably illustrated. Without knowing the author personally, the reviewer gets the impression that he is soundly conservative and open minded. Nevertheless, it is impossible for one urologist to review a book by another without discovering many differences of opinion.

The complete omission of discussions of operative technique (except for operative cystoscopy) should be mentioned, since the general practitioner at whom the book is avowedly aimed will need a text of operative surgery to accompany it. The omission was undoubtedly necessary to keep the book within one volume and is, on the whole, desirable.

Certain positive criticisms seem required. The legend that novocain is an effective topical anesthetic for the urethra ought to be destroyed. If it is, as the author says, "cruel to perform cystoscopy without analgesia," it is equally unfeeling to pretend that novocain, topically applied, produces any analgesia whatever.

Many will question Herman's statement that division of the isthmus of the horseshoe kidney is unsatisfactory; many more will properly deprecate his contention that only 15 per cent of hydronephroses due to obstruction at the ureteropelvic juncture are amenable to conservative surgery. Fifty per cent is more nearly correct.

The clinical picture of malignant renal neoplasms is rather inadequately presented in that its atypical clinical forms are insufficiently discussed.

All through the book the author exhibits a predilection for classifications so elaborate as to drive to despair any medical student who uses the book. This is especially striking in the case of "surgical inflammations of the kidney" (a poor term, incidentally, since it conveys the impression that the infections under discussion result from surgical operations), an outline classification of which occupies almost a whole page; nevertheless, the section is well done. Those who try to acidify infected urine with the recommended doses (15 to 60 gr. daily) of calcium or ammonium chloride are doomed to disappointment; it is doubtful whether methenamine is any longer regarded as "the most dependable drug." The newer antiseptics, including the ketogenic diet, are well discussed.

The section on the "neurogenic bladder" is entirely inadequate. Passing over the fact that the bladder cannot originate from the nervous system, an assumption certainly implied by the term, the discussion overlooks the recent literature and omits all reference to such important means of treating neurogenic vesical dysfunction as training, rest of the bladder, the use of pilocarpine to stimulate the detrusor, and the relief of complicating obstructions at the vesical neck. Measures for encouraging the development of an automatic bladder without catheterization in transverse myelitis are surely deserving of more emphasis. The author gives the im-

pression, I think unintentionally, that its appearance is an accident; actually its development can be brought about deliberately without catheterization.

The chapter on cystitis fails, in an otherwise adequate presentation, to mention the most useful symptomatic treatment in interstitial cystitis—overdistention under anesthesia.

The section on obstruction at the vesical neck is excellent despite its advocacy of the now outworn gradual decompression of the distended bladder which is surely only of historical interest. The author takes a rational view of the indications for the various methods of relief without attempting to maintain the superiority of one method in all circumstances as so many writers now attempt to do. One may justifiably wonder what he means by "maturity" of prostatic hypertrophy, and criticize his use of the term "radical prostatectomy" for suprapubic or perineal enucleation of hypertrophies. Many will question his belief that transurethral resection is more dangerous than prostatestomy in larger prostates and in older patients. Emphasis ought to be given to the value of transfusion of blood in shock following operations upon the prostate. Otherwise the section on care after transurethral resection is very good.

In discussing cancer of the prostate Dr. Herman brings out the fact, overlooked in several recent papers which deprecate transurethral operations, that a cancer concealed within a hypertrophied lobe is not often cured by enucleation of that lobe; he points out that arguments for radical perineal prostatectomy lose much of their force when one considers how infrequently the operation is feasible (10 per cent or less of the cases of cancer).

Despite the minor criticisms cited, this is a sound and valuable book and is extraordinarily complete for a single volume. It has another advantage over many of the current single volume texts in that it contains a bibliography which, while brief, affords a satisfactory entree into the literature for the reader of inquiring mind.

The International Medical Annual: A Year Book of Treatment and Practitioner's Index, Fifty-Sixth Year. Edited by H. Letheby Tidy and A. Rendle Short. Cloth. Pp. 615, with 68 plates. Baltimore, Md., 1938, William Wood and Company. $6.

This book is a review of the advances in the whole field of medicine during 1937. The pertinent literature in the various specialties is summarized in alphabetical order rather than by section. Among the reviewers of the surgical literature are Hamilton Bailey, Stanford Cade, A. Tudor Edwards, John Fraser, Ernest W. Hey Groves, C. Langton Hewer, J. P. Lockhart-Mummery, and A. Rendle Short.

In the introduction the editors give a survey of recent trends of medical practice. In the reviews that follow, one is impressed by the variety of subjects presented and the thoroughness with which they are discussed. The sections on the surgical treatment of bronchiectasis, thyroid diseases, and x-ray diagnosis are especially good. The review of the sulphonamides and the chemotherapy of bacterial invasion is one of the best written to date.

The printing and illustrations are excellent. The index is fairly comprehensive. In all, this is a well-written compilation and better than the average book of its kind.